DISSERTATION

ON

"A STUDY OF CLINICAL PROFILE OF FIVE HUNDRED CASES OF PRIMARY HEADACHES"

Submitted in partial fulfilment of

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF CLINICAL PROFILE OF FIVE HUNDRED CASES OF PRIMARY HEADACHES"** submitted by **Dr. C.SELVARAJ** appearing for **D.M. Neurology** Degree (Branch - I) examination in **August 2011** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, and India.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF CLINICAL PROFILE OF FIVE HUNDRED CASES OF PRIMARY HEADACHES"** is done by me at Government Rajaji Hospital, Madurai during 2008-2011 under the guidance and supervision of **Prof.DR.N.MUTHUVEERAN M.D., D.M.**

The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M. degree in Neurology.**

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CONTENTS

LIST OF ABBREVIATIONS

MWA – Migraine with Aura

MWOA – Migraine without Aura

MWWOA - Migraine with and without Aura

CM- Complications of Migraine

PM- Probable Migraine

TTH-Tension type Head ache

FETTH-Frequent Episodic Tension type Head ache

IETTH-Infrequent Episodic Tension type head ache

PTTH- Probable Tension type Head ache

CTTH-Chronic Tension type head ache

TAC- Trigeminal Autonomic Cephalagias

CCH – Chronic Cluster Headache

ECH –Episodic Cluster Headache

EPHC – Eposodic Paroxysmal Hemicrania

CPHC – Chronic Paroxysmal Hemicrania

SUNCT- Sudden Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing.

DFI-12m- Disease free interval for 12 months

DFI-6m+ - Disease free interval 6months -12 months

DFI-6m - Disease free interval less than 6 months

NR – No response

IHS – International Headache Society

Introduction

Headache is one among the most common medical complaints. Various forms of headache, properly called headache disorders, are among the most common disorders of the nervous system. They are pandemic and, in many cases, life-long conditions. Recurrent headache disorders impose a substantial burden on headache sufferers, family and society. Almost 90% headache cases seen in practice are classified as primary headaches, and they are defined as headaches that are not caused by an identifiable underlying structural, vascular or systemic illness. Although the epidemiology of headache disorders is only partly documented, taken together, headache disorders are extraordinarily common.This work of dissertation has been done with an aim at documenting the different types of primary headaches, their clinical presentations and responses to prophylaxis among patients presented to the Neurology Out Patient Department, Govt.Rajaji Hospital, Madurai during an one year period.

Aims and Objectives

This study is aimed to

1. To classify and study the incidence of various types of Primary Headache among patients presented at Neurology Outpatient Department, Govt. Rajaji Hospital, Madurai.
2. Study the Clinical Profile of each type of Primary Headache.
3. Study the various patterns of response to prophylaxis in Migraine.

Review of Literature

Headache or ***Cephalalgia*** is a symptom of a number of different conditions of the head and neck, caused by benign or at times a medical emergency. It ranks among the most common pain complaints. One in three persons suffers a severe headache at some point in life.

History:

The earliest references date back to a papyrus (ancient medical text book) dating as old as 3500 BC in the tomb of Thebes's mentions that the king in the tomb had suffered all his life of a sickness of half head. Headache with neuralgia is referred to since the times of ancient Egyptians (1200 BC), with Hippocrates (460-377BC) describing the visual aura in migraine and its relief through vomiting[1]. **Aretaeus of Cappadocia,** in the2nd century described a unilateral headache associated with vomiting, with headache-free intervals is credited as the "discoverer" of migraine[2].**Galenus of Pergamon** used the term "hemicrania" (half-head) and thought there was a connection between the stomach and the brain, as nausea and vomiting often accompanied an attack[2].In the Medieval Ages migraine was recognized as a disorder that was treated with hot irons, blood letting and even witchcraft. *Bibliotheca Anatomica, Medic, Chirurgica*, published in London (1712), describes five major types of headaches, including "Megrim", recognized later as classic migraine[2]. Graham and Wolffe (1938) advocated ergotamine tart for

relieving migraine. Harold Wolffe (1950) developed the experimental approach to the study of headache and elaborated the vascular theory of migraine[2]. Harold Wolffe (1950), enunciated a list of pain sensitive structures.[3,4]

There are references to a number of different classification systems for headaches with a rich history. The first recorded system that resembles the modern one was published by Thomas Willis, in *De Cephalagia* in 1672[4]. In 1787 Christian Baur classified headaches into idiopathic (primary headaches) and symptomatic (secondary headaches) and defined 84 categories. NIH developed a classification system in 1962[5].

Demographics & Etiology:

Epidemiology: According to IHS, migraine constitutes 16% of primary headaches. Migraine afflicts 10-20% of the general population. Worldwide 15-20% of women and 10-15% of men suffer from migraine. In India, 15-20% of people suffer from migraine with an adult female: male ratio of 2:1.[5] In childhood migraine, boys and girls are affected equally until puberty. In individuals older than 12 years, the prevalence increases in both males and females, and the incidence declines in individuals older than 40 years, except for women in perimenopause. The overall prevalence is higher in females than in males. The female-to-male ratio increases from 2.5:1 at puberty to 3.5:1 at age 40 years, after which it declines. The incidence of migraine with aura

peaks in boys at around age 5 years and in girls at around age 12-13 years. The incidence of migraine without aura peaks in boys at age 10-11 years and in girls at age 14-17 years. The incidence of migraine in females of reproductive age has increased over the last 20 years, probably due to more awareness of the condition.

Age-Gender Incidence: Migraine is an extremely common condition which will affect 12–28% of people at some point in their lives. The one year prevalence of migraine ranges from 6–15% in adult men and from 14–35% in adult women[6]. These figures vary substantially with age: approximately 4–5% of children aged under 12 suffer from migraine, with little apparent difference between boys and girls.

Pathophysiology of Migraine:

Vascular theory: Migraines can begin when blood vessels in the brain contract and expand inappropriately. This may start in the occipital lobe, in the back of the brain, as arteries undergo spasm. The reduced flow of blood from the occipital lobe triggers the visual aura[7].Wolffe et al (1940)[3] believed that intracranial vasoconstriction is responsible for the aura of migraine and that subsequent rebound vasodilatation and activation of perivascular nociceptive nerves resulted in headache, based on the observations that (1) extracranial vessels become distended and pulsatile during a migraine attack, (2) stimulation of intracranial vessels in an awake person induces headache,

and (3) vasoconstrictors (e.g. ergots) improve the headache, whereas vasodilators (e.g. nitroglycerin) provoke an attack.[8]This theory has fallen out of favour, migraine is now considered due to neuronal dysfunction while the vascular changes being secondary to neuronal dysfunction.

Depolarization theory: A phenomenon known as cortical spreading depression can cause migraines.[9]In cortical spreading depression, neurological activity is depressed over an area of the cortex of the brain. This situation results in the release of inflammatory mediators leading to irritation of cranial nerve roots, most particularly the trigeminal nerve, which conveys the sensory information for the face and much of the head. A spreading depolarization may begin 24 hours before the attack, with onset of the headache occurring around the time when the largest area of the brain is depolarized. A French study in 2007, using the PET technique identified the hypothalamus as being critically involved in the early stages.[10]

Cortical spreading depression: In 1944, Leao proposed this theory to explain the mechanism of migraine with aura. A migraine aura is due to well-defined wave of neuronal excitation in the cortical gray matter that spreads from its site of origin at the rate of 2-6 mm/min; followed by a wave of neuronal suppression in a similar fashion. The blood vessels in the area simultaneously dilate and constrict. Hence, migraine aura is considered a cortical event with definite and well-defined neuroelectrical basis. The

neurochemical basis of the CSD is the release of potassium or the excitatory amino acid glutamate from neural tissue. This release depolarizes the adjacent tissue, which, in turn, releases more neurotransmitters, propagating the spreading depression. Positron emission tomography (PET) scanning demonstrates that blood flow is moderately reduced during a migrainous aura, but the spreading oligemia does not correspond to vascular territories. Specific groups of patients with migraine have a genetic defect leading to a lowered threshold for CSD, and this is called familial hemiplegic migraine (FHM). However, for the vast majority of patients, a clear metabolic or genetic defect that easily explains this neuronal excitability cannot be determined.[11]

Other theories: Another theories propose that abnormalities in neurotransmitters like serotonin,dopamine and deficiency of magnesium.

Clinical Features:

The four phases of a migraine attack listed below are common but not necessarily experienced by all migraine sufferers. Additionally, the phases experienced and the symptoms experienced during them can vary from one migraine attack to another.

Prodrome phase: Prodromal symptoms occur in 40–60% of migraineurs (migraine sufferers). This phase may consist of altered mood, irritability, depression or euphoria, fatigue, yawning, excessive sleepiness, craving for

certain food (e.g. chocolate), stiff muscles (especially in the neck), constipation or diarrhea, increased urination, and other visceral symptoms[12].These symptoms usually precede the headache phase of the migraine attack by several hours or days, and experience teaches the patient or observant family how to detect that a migraine attack is near.

Aura phase: For the 20–30% of individuals who suffer migraine with aura, this aura comprises focal neurological phenomena that precede or accompany the attack. They appear gradually over 5 to 20 minutes and generally last fewer than 60 minutes. The headache phase of the migraine attack usually begins within 60 minutes of the end of the aura phase, but it is sometimes delayed up to several hours, and it can be missing entirely. Symptoms of migraine aura can be visual, sensory, or motor in nature[13]. Visual aura is the most common of the neurological events. There is a disturbance of vision consisting usually of unformed flashes of white and/or black or rarely of multicolored lights (photopsia) or formations of dazzling zigzag lines (scintillating scotoma; often arranged like the battlements of a castle, hence the alternative terms "fortification spectra" or "teichopsia". The somatosensory aura of migraine consists of feeling of pins-and-needles experienced in the hand and arm as well as in the nose-mouth area on the same side. [14]

Pain phase: The typical migraine headache is unilateral, throbbing, moderate to severe and can be aggravated by physical activity. Not all of these features are necessary. The pain may be bilateral at the onset or start on one side and become generalized, and usually alternates sides from one attack to the next. The onset is usually gradual. The pain peaks and then subsides, and usually lasts between 4 and 72 hours in adults and 1 and 48 hours in children. The frequency of attacks is extremely variable. The head pain varies greatly in intensity. The pain of migraine is invariably accompanied by other features. Many patients experience sensory hyperexcitability manifested by photophobia, phonophobia and seek a dark and quiet room. Blurred vision, nasal stuffiness, diarrhea, polyuria, pallor or sweating may be noted during the headache phase. There may be localized edema of the scalp or face, scalp tenderness, prominence of a vein or artery in the temple, or stiffness and tenderness of the neck

Postdrome phase: The patient may feel tired, have head pain, cognitive difficulties, "hungover", gastrointestinal symptoms, mood changes and weakness.[15]

Defining Pain Severity: The IHS defines the intensity of pain with a verbal, four-point scale.[17]

Number	Name	Annotations
0	No pain	
1	Mild pain	Does not interfere with usual activities
2	Moderate pain	Inhibits, but does not wholly prevent usual activities
3	Severe pain	Prevents all activities

Triggers: A migraine trigger is any factor that, on exposure or withdrawal, leads to the development of an acute migraine headache. Triggers may be categorized as behavioural, environmental, infectious, dietary, chemical, or hormonal. Migraine attacks may be triggered by allergic reactions, bright lights, loud noises, and certain odours or perfumes, head trauma, physical or emotional stress, changes in sleep patterns, smoking, skipping meals, alcohol, menstrual cycle fluctuations, foods containing tyramine, monosodium glutamate (MSG) or nitrates, other foods such as chocolate, dairy products, and fermented or pickled foods, medications (eg, nitroglycerin, histamine, reserpine, hydralazine, ranitidine, estrogen).[17] One study found that for some migraineurs in India, washing hair in a bath was a migraine trigger. The triggering effect was probably due to delayed drying of the wet hair.[18]

Diagnostic Procedures:

Neuroimaging is indicated for any of the following: [A] First or worst headache of the patient's life [B] Change in frequency, severity, or clinical features of the headache [C] Abnormal neurological examination [D] Progressive or new daily persistent headache [E] Neurological symptoms that do not meet the criteria for migraine with typical aura or that themselves warrant investigation [E] Persistent neurologic deficit [F] Hemicrania that is always on the same side and associated with contralateral neurological symptoms [G] Inadequate response to routine therapy [H] Atypical clinical presentation. Neuroimaging studies that may be appropriate include CT scan and MRI. Other studies such as angiography, MRA, and MRV also may be indicated.[10]

Electrophysiology: The electroencephalogram (EEG) was a standard test for evaluation of headaches in the pre-CT scan era. Gronseth and Greenberg [38] reviewed the literature from 1941 to 1994 on the usefulness of EEG in the evaluation of patients who had headache. Most of the articles had serious methodological flaws. The only significant abnormality reported in studies with a relatively nonflawed design was prominent driving in response to photic stimulation in migraineurs [9].This finding, although interesting, is not necessary for the clinical diagnosis of migraine.

Autonomic Function Tests: The rationale to study functions of the ANS in headache is primarily based on clinical observations. Changes of ANS functions are obvious in cluster headache with autonomic symptoms[19] and have been extensively investigated for these pains in the last 20 years. Changes of ANS function are suspected to be important in migraine but are less obvious.

Treatment:

Conventional treatment focuses on three areas: trigger avoidance, abortive therapy, and prophylaxis. Patients who experience migraines often find that the recommended migraine treatments are not 100% effective at preventing migraines, and sometimes may not be effective at all. Drug therapy is considered *effective* to reduce the frequency or severity of migraine attacks by 50%.[20] Children and adolescents are often first given drug treatment, but the value of diet modification should not be overlooked.

I. Abortive Therapy: The abortive treatment of migraine is divided into (1) Symptomatic treatment (2) Specific Anti-migraine drugs.

Non Specific Treatment of Migraine: There is a large group nonspecific acute medications for migraine, that include nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin (ASA), acetaminophen (APAP), analgesic combinations with or without caffeine, opioids, butalbital, isometheptene, also often in combinations, and medication classes including

antihistamines, antinauseants, antiepilepsy drugs, and muscle relaxants. Aspirin-acetaminophen-caffeine (AAC) mixtures are Food and Drug Administration approved for migraine based on a randomized controlled trial. NSAIDs, such as naproxen, diclofenac and solubilized ibuprofen, also are superior to placebo in RCTs. Evidence for opioid use in acute migraine generally is negative.

Specific Anti Migraine Treatment: (1) Triptans are serotonin -1B/D (5HT1B/D) agonists that work via the serotonin-1D receptors to inhibit CGRP and inflammatory peptide release in the meninges and prevent the pain signal from returning from the periphery to the trigeminal nucleus caudalis. They work via the 5HT1B receptor to constrict vessels dilated by CGRP. Because there are some 5HT1B receptors in coronary and other arteries, triptans are contraindicated in patients who have vascular disease. Triptans are classified into Group I and II. Group-I triptans are fast acting with response rates of two hours of medication eg- Sumatriptan, Zolmitriptan, Rizatriptan, Almotriptan and Eletriptan. While group II triptans have a minimally slower rate of activity at 4 hours eg: Naratriptan and Frovatriptan. Selecting the triptan necessitates determining how fast a migraine worsens. If onset is quick, a group I triptan is necessary. If emesis is common, a non oral formulation like sumatriptan SC or Zolimitriptan Nasal spray are preferred. Some evidence suggests a triptan-NSAID combination can reduce headache recurrence.

(2) Ergots: Until the introduction of sumatriptan in 1991, ergot derivatives were the primary oral drugs available to abort a migraine once it is established. Ergot drugs can be used either as a preventive or abortive therapy, though their relative expense and cumulative side effects suggest reserving them as an abortive rescue medicine. However, ergotamine tartrate tablets (usually with caffeine), though highly effective, and long lasting (unlike triptans), have fallen out of favour due to the problem of ergotism.[21] Both ergot and triptans have the same risk for vasoconstriction DHE is tolerated far better, is useful as an acute treatment of long migraines because of low recurrence, and can be used repetitively for status migrainosus and in detoxification from rebound. According to AAN guidelines, maximum dosing for DHE is 3 mg per day, up to 21 mg per week. Intranasal DHE dosing requires one spray (0.5 mg) into each nostril (without sniffing) at the first sign of migraine, followed 15 minutes later by an additional spray into each[22]

Other agents: Steroids: Based on a recent meta analysis a single dose of IV dexamethasone, when added to standard treatment, is associated with a 26% decrease in headache recurrence.[23] Recently it has been found that calcitonin gene related peptides (CGRPs) play a role in the pathogenesis of the pain associated with migraine as triptans also decrease its release and action. CGRP receptor antagonists such as olcegepant and telcagepant are being investigated both in vitro and in clinical studies for the treatment of migraine.

II. Prophylactic Therapy

US evidence-based guidelines for preventive treatment of migraine include the following:

1. Recurring migraine that significantly interferes with the patient's daily routine despite acute treatment (eg, two or more attacks a month that produce disability that lasts 3 or more days, headache attacks that are infrequent but produce profound disability
2. Failure of, contraindication to, or troublesome side effects from acute medications
3. Overuse of acute medications
4. Special circumstances, such as hemiplegic migraine or attacks with a risk for permanent neurologic injury.
5. Frequent headaches (more than two a week) or a pattern of increasing attacks over time, with the risk for developing medication overuse headache.
6. Patient preference, that is, the desire to have as few acute attacks as possible

The major medication groups for preventive migraine treatment include

1. Anticonvulsants
2. Antidepressants
3. Beta-adrenergic blockers
4. Calcium channel antagonists

5. Serotonin antagonists
6. Botulinum neurotoxins
7. Nonsteroidal anti-inflammatory drugs
8. Others - Riboflavin, Magnesium.

Preventive treatment is often recommended for only 6 to 9 months; however, to date, no randomized placebo-controlled trials have been performed to investigate migraine frequency after the preventive treatment has been discontinued.

Beta-Adrenergic Blockers: Beta—blockers, the most widely used class of drugs in prophylactic migraine treatment, are approximately 50% effective in producing a greater than 50% reduction in attack frequency. Evidence has consistently demonstrated that non selective beta-blocker propranolol is significantly effective, selective b1-blockers metoprolol, atenolol, bisoprolol, nadolol, and timolol are also effective, while beta-blockers with intrinsic sympathomimetic activity (eg, acebutolol, alprenolol, oxprenolol, pindolol) are not effective for migraine prevention. Propranolol is effective for migraine prevention at a daily dose of 120 to 240 mg, but no correlation has been found between its dose and its clinical efficacy.[22] The action of beta—blockers is probably central and could be mediated by (1) inhibiting central b-receptors that interfere with the vigilance-enhancing adrenergic pathways, (2) interaction with 5—HT receptors (but not all effective beta—blockers bind to the 5—HT receptors), and (3) cross-

modulation of the serotonin system. Propranolol inhibits nitric oxide (NO) production by blocking inducible NO synthase. Propranolol also inhibits kainate-induced currents and is synergistic with N-methyl- D-aspartate blockers, which reduce neuronal activity and have membrane-stabilizing properties[23]

Antidepressants: Antidepressants consist of several different drug classes with different mechanisms of action. Only one member of the class of tricyclic antidepressants, Amitriptyline has proved efficacy in migraine. Although the mechanism by which antidepressants work to prevent migraine headache is uncertain, it does not result from treating masked depression. Antidepressants are useful in treating many chronic pain states, including headache, independent of the presence of depression, and the response occurs sooner than the expected antidepressant effect. The antidepressants that are clinically effective in headache prevention inhibit noradrenaline and 5—HT reuptake or are antagonists at the 5—HT2 receptors. The TCA dose range is wide and must be individualized. Most TCAs are sedating.

Calcium Channel Antagonists: The mechanism of action of the calcium channel antagonists in migraine prevention is uncertain, but possibilities include inhibition of 5—HT release, neurovascular inflammation, or the initiation and propagation of cortical spreading depression. *Flunarizine*, a nonselective calcium channel antagonist with antidopaminergic properties, was superior to placebo in six of seven randomized clinical trials.[24] The dose

is 5 to 10 mg given at night, women seem to need lower doses than men. The most prominent AEs include weight gain, somnolence, dry mouth, dizziness, hypotension, occasional extrapyramidal reactions, and exacerbation of depression. Because of its side effects, flunarizine should be considered as a second-line drug for migraine prevention, after beta-blockers. *Verapamil* was more effective than placebo in two of three trials, but both positive trials were small and dropout rates were high, rendering the findings uncertain. Rigorous randomized controlled trial evidence does not exist to support the use of verapamil for migraine. Nimodipine, nicardipine, diltiazem, and cyclandelate, other nonselective calcium channel antagonists, cannot be recommended for migraine prophylaxis.[24]

Anticonvulsants: Anticonvulsants are increasingly recommended for migraine prevention because of well-conducted placebo-controlled trials. With the exception of valproic acid, topiramate, and zonisamide, anticonvulsants may substantially interfere with the efficacy of oral contraceptives. ***Carbamazepine*** 600 to 1200 mg/d, may be effective in preventive migraine treatment but it is rarely used in clinical practice for this purpose.[36] ***Gabapentin*** (1800–2400 mg) showed efficacy in a placebo-controlled double-blind trial, the attack frequency was reduced by 50% in approximately one third of patients. ***Valproic Acid*** is a simple 8-carbon, 2-chain fatty acid. Divalproex sodium is a combination of valproic acid and sodium valproate. Both are effective as is an extended-release form of

divalproex sodium. In 1992, Hering and Kuritzky evaluated the efficacy of sodium valproate in migraine treatment in a double-blind, randomized, crossover study. Nausea, vomiting, and gastrointestinal distress are the AEs that occur most commonly; their incidence decreases, however, particularly after 6 months. Later, tremor and alopecia can occur. Valproate has little effect on cognitive functions and rarely causes sedation.[24] ***Topiramate****:* Two large, pivotal, multicenter, randomized, double-blind, placebo-controlled clinical trials assessed the efficacy and safety of topiramate (50, 100, and 200 mg/d) in migraine prevention[24].The most common AE of topiramate is paresthesia; other common AEs are fatigue, decreased appetite, nausea, diarrhea, weight loss, taste perversion, hypoesthesia, and abdominal pain. The most common central nervous system AEs were somnolence, insomnia, mood problems, anxiety, difficulty with memory, language problems, and difficulty with concentration. Renal calculi can occur with topiramate use.

Tension-type headache (TTH)

Tension headaches, renamed **tension-type headaches** by the International Headache Society in 1988, are the most common type of primary headaches. Tension-type headaches account for nearly 90% of all headaches. Approximately 3% of the population suffers from chronic-tension type headache. Tension headaches were previously termed as - muscle contraction headache, psychomyogenic headache, stress headache, ordinary headache, essential headache, idiopathic headache and psychogenic headache.

The chronic subtype is a serious disease causing greatly decreased quality of life and high disability. The exact mechanisms of tension-type headache are not known.

Pathophysiology:

Various precipitating factors may cause TTH in susceptible individuals [2]. One half of patients with TTH identify stress or hunger as a precipitating factor that include - Stress - Usually occurs in the afternoon after long stressful work hours, Sleep deprivation & Eyestrain, Uncomfortable stressful position and/or bad posture, Irregular meal time (hunger), and Caffeine withdrawal. Until recently it was believed that tension headaches were caused by muscle tension around the head and neck. Recent research has shown that tension headache patients do not have increased muscle tension.[25] Another theory is that the pain may be caused by a malfunctioning pain filter which is located in the brain stem. The view is that the brain misinterprets information, for example from the temporal muscle or other muscles, and interprets this signal as pain. The sensitization of pain pathways may be caused by or associated with activation of nitric oxide synthase (NOS) and the generation of NO. Patients with chronic tension-type headache have increased muscle and skin pain sensitivity, demonstrated by low mechanical, thermal and electrical pain thresholds. Hyperexcitability of central nociceptive neurons (in trigeminal spinal nucleus, thalamus and cerebral

cortex) is believed to be involved in the pathophysiology of chronic tension-type headache.[26]

Frequency: Tension-type headaches can be episodic or chronic. Episodic tension-type headaches are defined as tension-type headaches occurring fewer than 15 days a month, whereas chronic tension headaches occur 15 days or more a month for at least 6 months. Tension-type headaches can last from minutes to days, months or even years, though a typical tension headache lasts 4–6 hours

Clinical Features:

Pain onset in tension-type headache can have a throbbing quality and is usually more gradual than onset in migraines. Compared with migraines, tension-type headaches are more variable in duration, more constant in quality, and less severe. Their features are as follows

- Duration of 30 minutes to 7 days
- No nausea or vomiting (anorexia may occur)
- Photophobia and/or phonophobia
- Minimum of 10 previous headache episodes; fewer than 180 days per year with headache to be considered "infrequent"
- Bilateral and occipitonuchal or bifrontal pain - Pain described as "fullness, tightness/squeezing, pressure," or "bandlike/viselike"

- May occur acutely under emotional distress or intense worry & Insomnia, often present upon rising or shortly thereafter
- Muscular tightness or stiffness in neck, occipital, and frontal regions
- Duration of more than 5 years in 75% of patients with chronic headaches
- Difficulty concentrating
- No prodrome

Physical examination serves mainly to exclude the possibility of other headache causes.

- Vital signs should be normal.
- Normal neurologic examination
- Tenderness may be elicited in the scalp or neck, but no other positive physical exam findings should be noted.
- Pain should not be elicited over temporal arteries or positive trigger zones.
- Some patients with occipital tension headaches may be very tender when upper cervical muscles are palpated.
- Pain associated with neck flexion and stretching of paracervical muscles must be distinguished from nuchal rigidity associated with meningeal irritation.

Laboratory Studies:

Laboratory work should be unremarkable in cases of tension-type headache. Specific tests should be obtained if the history or physical examination suggests another diagnostic possibility. Head CT scan or MRI is necessary only when the headache pattern has changed recently, the headache cannot be clearly defined by the clinician as a common primary headache disorder or neurologic examination reveals abnormal findings.

Treatment:

Nonsteroidal anti-inflammatory drugs (NSAIDs): These agents may alleviate headache pain by inhibiting prostaglandin synthesis, reducing serotonin release, and blocking platelet aggregation. Although the effects of NSAIDs in the treatment of headache pain tend to be patient specific, ibuprofen is usually the drug of choice for initial therapy. Other options include naproxen, ketoprofen, and ketorolac.[27]

Physical therapy for patients with headache includes warm and cold packs, ultrasound, and electrical stimulation. Regular exercise, stretching, balanced meals, and adequate sleep is part of a headache prevention program. Trigger point injections, occipital nerve blocks, or changes that improve posture may be used.

Deterrence/Prevention: Deterrence and prevention of headache may include the following: Physical therapy, Biofeedback and relaxation therapy, Cervical traction and Injection of trigger points.

Complications: Complications of headache may include the following: Undue reliance on nonprescription caffeine-containing analgesics, Dependence on/addiction to narcotic analgesics, GI bleed from use of NSAIDs, Risk of epilepsy 4 times greater than that of the general population

Prognosis: Headache may become chronic if life stressors are not changed. Most cases are intermittent and do not interfere with work or normal life span.

Trigeminal autonomic cephalalgias (TAC)

Cluster headache is a neurological disease that involves, as its most prominent feature, an immense degree of pain. "Cluster" refers to the tendency of these headaches to occur periodically, with active periods interrupted by spontaneous remissions. Cluster headaches have been called by several other names in the past including Erythroprosopalgia of Bing, Ciliary neuralgia, Erythromelagia of the head, Horton's headache (named after Bayard T. Horton, an American neurologist), Histaminic cephalalgia, Petrosal neuralgia, sphenopalatine neuralgia, Vidian neuralgia, Sluder's neuralgia, and Hemicrania angioparalyticia[28]. It affects approximately 0.1% of the population, and men are more commonly affected than women. The

trigeminal autonomic cephalalgias share the clinical features of headache and prominent cranial parasympathetic autonomic features.

Epidemiology:

Cluster headaches are diagnosed more often in men. The male-to-female ratio in cluster headache ranges from 4:1 to 7:1. It primarily occurs between the ages of 20 to 50 years.[29]

Pathophysiology:

Cluster headaches are caused by the dilation of blood vessels which creates pressure on the trigeminal nerve. While this process is the immediate cause of the pain, the aetiology is not fully understood.

Among the most widely accepted theories is that cluster headaches are due to an abnormality in the hypothalamus.[29]Experimental and human functional imaging suggests that these syndromes activate a normal human trigeminal-parasympathetic reflex with clinical signs of cranial sympathetic dysfunction being secondary.

Genetics: There is a genetic component to cluster headaches, although no single gene has been identified as the cause. First-degree relatives of sufferers are more likely to have the condition than the population at large.

Triggers: Nitroglycerin (glyceryl trinitrate) can sometimes induce cluster headaches in sufferers in a manner similar to spontaneous attacks. Ingestion of alcohol, chocolate, exposure to hydrocarbons (petroleum solvents, perfume), heat, napping, nicotine may trigger cluster headaches.[30]

Clinical Features:

Cluster headaches are excruciating unilateral headaches of extreme intensity. The duration of the common attack ranges from as short as 15 minutes to three hours or more. The onset of an attack is rapid, and most often without the preliminary signs that are characteristic of a migraine. However, some sufferers report preliminary sensations of pain in the general area of attack, often referred to as "shadows", that may warn them an attack is imminent.[29] Though the headaches are almost exclusively unilateral, there are many documented as cases of "side-shifting" between cluster periods, or, even rarer, simultaneously (within the same cluster period) bilateral headache Trigeminal neuralgia can also bring on headaches with similar qualities. However in Trigeminal neuralgia the pain is mostly located around the "cheek" area and is described as being more lance-like in quality. [29]

Pain: The degree of pain involved in cluster headaches is markedly greater than in other headache conditions, including severe migraines, and experts believe that it may be the most severe pain known to medical science. It has been described by female patients as being more severe than childbirth.[29] The McGill Pain Index can be used to rate levels of pain. The pain is lancinating or boring in quality, and is located behind the eye or in the temple, sometimes radiating to the neck or shoulder. Analogies frequently used to describe the pain are a red-hot poker inserted into the eye, or a spike

penetrating from the top of the head, behind one eye, radiating down to the neck, or sometimes having a leg amputated without any anaesthetic.

Cyclical recurrence and regular timing: Cluster headaches are occasionally referred to as "alarm clock headaches", because of its ability to wake a person from sleep, and because of the regularity of its timing in that both the individual attacks and the clusters themselves can have a regularity; attacks striking at a precise time of day each morning or night is typical, even precisely at the same time a week later.[29]

Other symptoms include ptosis, conjunctival injection, lacrimation, rhinorrhea, facial blushing, swelling and sweating. These features are known autonomic symptoms. The attack is also associated with restlessness, aversion to bright lights and loud noise during the attack, nausea is comparatively rare.

Episodic or Chronic Cluster Headache: occur once or more daily, often at the same times each day, for a period of several weeks, followed by a headache-free period lasting weeks, months, or years. Cluster headaches occurring in two or more cluster periods lasting from 7 to 365 days with a pain-free remission of one month or longer between the clusters are considered episodic. If the attacks occur for more than a year without a pain-free remission of at least one month, the condition is considered chronic. Chronic clusters run continuously without any "remission" periods between cycles. The condition may change from chronic to episodic and from episodic

to chronic. Remission periods lasting for decades before the resumption of clusters are known to occur.

Treatment:

Medications to treat cluster headaches are classified as either abortives or prophylactics. In addition, short-term transitional medications (such as steroids) may be used while prophylactic treatment is instituted and adjusted. With abortive treatments often only decreasing the duration of the headache and preventing it from reaching its peak rather than eliminating it entirely, preventive treatment is always indicated for cluster headaches, to be started at the first sign of a new cluster cycle.

Abortive treatment: During the onset of a cluster headache, some patients respond to rapid inhalation of pure oxygen (12-15 litres per minute in a non-rebreathing mask).[30] When used at the onset this can abort the attack in as little as 1 minute or as long as 10 minutes. Once an attack is at its peak, oxygen therapy appears to have little effect.

Alternative first-line treatment is subcutaneous administration of triptan drugs, like sumatriptan and zolmitriptan. The injectable form of sumatriptan has been shown to abort a cluster headache within fifteen minutes in 96% of cases. Because of the rapid onset of an attack, the triptan drugs are usually taken by subcutaneous injection rather than by mouth. Beta blockers as a treatment has been tried. Lidocaine and other topical anesthetics

sprayed into the nasal cavity may relieve or stop the pain. Vaso-constrictors such as ergot compounds were also used. Other abortive remedies that work include ice, hot showers, cool or lukewarm water sprayed on the face around the sinus, temple, and ear areas, breathing cold air, caffeine, and drinking large amounts of water in the early stages of an attack. Hyperbaric oxygen therapy has been used successfully in treating cluster headaches though it was not shown to be more successful than surface oxygen[30].

Prophylactic Treatment: A wide variety of prophylactic medicines are in use, and patient response to these is highly variable. Current European guidelines suggest the use of the calcium channel blocker verapamilate at a dose of at least 240 mg daily. Steroids, such as prednisolone, are also effective, with a high dose given for the first five days or longer before tapering down. Methysergide, lithium and the anticonvulsant to piramate are recommended as alternative treatments. Muscle relaxants and atypical anti-psychotics have also been used. Magnesium supplements have been shown to be of some benefit in about 40% of patients. Melatonin has also been demonstrated to bring significant improvement in approximately half of episodic patients. Other neuropathic pain alleviating agents such as the Tricyclic antidepressants including Amitriptyline and Nortriptyline can also be used.[30]

Materials and Methods

The patients registered at Neurology Outpatient Department, Govt. Rajaji Hospital, Madurai during a one year period between the March 2009 and February 2010 were taken for this study. The clinical material was collected from the records and by patient interviews with a detailed pre-prepared proforma [Appendix-1]. Antimigraine prophylactic drugs were given to patients based on their age, sex and comorbidities.

Inclusion Criteria:

1. Patients registered at the Neurology Outpatient Department, Govt. Rajaji Hospital, and Madurai during an one year period between the March 2009 and February 2010.
2. Patients in all age groups in both sexes, of any racial and socioeconomic denomination and profession were included
3. Patients were followed up for one year from the date of their registration and those who completed one year of follow up were included. All headaches were classified according to the International Headache Society Criteria 2004.

Exclusion Criteria:

1. Patients with systemic, metabolic, traumatic disorders and or radiological findings that were documented to be directly or indirectly related to the causation of headache were excluded.

2. Headache located or transmitted to the Cranium from the Maxillo / Mandibulofacial region, pharynx, paranasal sinuses, neck and ear were excluded.
3. Patients with incomplete clinical profiles, diagnostic & treatment records were excluded.
4. Patients who did not complete the one year follow up were excluded.

Evaluation of Results:

The clinical presentations, radiological features, laboratory results, neurophysiological patterns, treatment and its response on follow up, the progress and complications were documented and tabulated in a Master Chart (Appendix-2). The various parameters of the patients were compared, classified and analysed with specific reference to national and international studies.

RESULTS

A total of 502 patients registered at the Headache OPD, Department of Neurology, Govt. Rajaji Hospital, Madurai Medical College, Madurai between 1st March 2009 and 28th February 2010 were included in the study. Of the total of 502 patients, 382 (76.09%) patients had migraine, 100 (19.92%) had tension type headache, 12 (2.39%) Trigeminal autonomic cephalagia, 8 (1.59%) had other types of primary headaches.

No	Type of Primary Headache	No of Patients	Percentage
1	Migraine	382	76.09
2	Tension Type Headache	100	19.92
3	Trigeminal autonomic cephalalgia	12	2.39
4	Other Primary headaches	8	1.59
	TOTAL NO CASES	502	

Classification of Headaches

Of the 382 case of Migraine, 186(48.69%) patients had Migraine without Aura, 36 (9.42%) patients had Migraine with Aura, 90(23.56%) patients had Migraine which presented with & without aura, 62(16.23%) patients had complications of migraine and 8(2.09%) patients had probable migraine.

No	Type of Migraine	IHS Code	No of Patients
1	Migraine without Aura	1.1	186
2	Migraine with Aura	1.2	36
3	Migraine with and without aura	1.1 / 1.2	90
4	Complications of Migraine	1.5	62
5	Probable Migraine	1.6	8
	TOTAL		382

Of the patients Migraine with Aura (36 patients) the predominant subtype was Typical Aura with Headache (16 patients). Our study also included 6 patients of Typical Aura with Non Migraine HA, 4 patients with Typical Aura without Headache and 10 patients with Basilar Type Migraine. Our study group did not have patients with Familial Hemiplegic Migraine and Sporadic Hemiplegic Migraine.

No	Type of Migraine with Aura	IHS Code	No of Patients with Migraine with Aura
1	Typical Aura with Headache	1.2.1	16
2	Typical Aura with Non Migraine HA	1.2.2	6
3	Typical Aura without Headache	1.2.3	4
4	Familial Hemiplegic Migraine	1.2.4	0
5	Sporadic Hemiplegic Migraine	1.2.5	0
6	Basilar Type Migraine	1.2.6	10
	TOTAL		36

Of the 62 patients with complications of migraine, 48 patients had Chronic Migraine (12 patients with Chronic Migraine since Onset & 36 patients with Episodic Headache converting to Chronic Migraine), 1 patient had Migrainous Infarction (Right Occipital Infarction) and 13 patients had Migraine Trigerred Seizures (Migraine Terminating as Seizures – 9, Migralepsy-4)

No	Type of Complications of Migraine	IHS Code	Sub Types	No of Patients	Total No of Patients
1	Chronic Migraine	1.5.1	Chronic Migraine since Onset	12	48
			Episodic Headache converting to Migraine	36	
2	Status Migrainosus	1.5.2			0
3	Persistent Aura without Infarction	1.5.3			0
4	Migrainous Infarction	1.5.4	Right Occipital Infarction	1	1
5	Migraine Triggered Seizures	1.5.5	Migraine Terminating as Seizures	9	13
			Migralepsy	4	
	TOTAL				62

In our study, we came across 52 patients with Migraine and Seizures, 13 patients had Migraine Triggered Seizures, 17 patients had Post ictal migrainous headache and 22 patients had Migraine & Coexistent Seizure. Of the 13 patients who had migraine triggered seizures 9 patients had migraine terminating as seizures and 4 patients had Migralepsy.

No	Migraine & Seizures	No of Patients
1	Migraine Triggered Seizures	13
2	Post Ictal Migrainous Headache	17
3	Migraine & Coexistent Seizure	22

Out of a total of 502 patients registered at the OPD, 100 (19.92%) had tension type headache, of which 16 patients had Infrequent Episodic TTH, 24 patients had Frequent Episodic TTH, 48 patients had Chronic TTH, and 12 patients had Probable TTH.

No	Type of TTH	IHS Code	No of Patients
1	Infrequent Episodic TTH	2.1	16
2	Frequent Episodic TTH	2.2	24
3	Chronic TTH	2.3	48
4	Probable TTH	2.4	12
	TOTAL		100

Out of a total of 502 patients registered at the OPD, 12 (2.39%) had Trigeminal autonomic cephalagia of which 6 patients had Cluster Headache, 5 patients had Paroxysmal hemicrania, 1 patients had Short

Lasting Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing.

No	Type of Trigeminal Autonomic Cephalalgia	IHS Code	No of Patients
1	Cluster Headache	3.1	6
2	Paroxysmal hemicrania	3.2	5
3	SUNCT	3.3	1
4	Probable TAC	3.4	0
	TOTAL		12

Of the 6 patients who had Cluster Headache 4 were classified as Episodic Cluster Headache and 2 as Chronic Cluster Headache. Of the 5 patients who had Paroxysmal Hemicrania, 3 were classified as Episodic Paroxysmal Hemicrania and 2 as Chronic Paroxysmal Hemicrania.

No	Type of Cluster Headache	IHS Code	No of Patients
1	Episodic Cluster Headache	3.1.1	4
2	Chronic Cluster Headache	3.1.2	2
	TOTAL		6

No	Type of TAC	IHS Code	No of Patients
1	Episodic Paroxysmal Hemicrania	3.2.1	3
2	Chronic Paroxysmal Hemicrania	3.2.2	2
	TOTAL		5

Other Primary headaches were few among the registered patients at the Neurology OPD. 8(1.59%) patients had other primary headaches, of which 2 patients had Primary Stabbing Headache, 1 patient had Primary Headache associated with Sexual Activity, 1 patient had Primary Hypnic Headache, and 4 patients had New Daily Persistent Headache.

No	Other Primary Headaches	IHS Code	No of Patients
1	Primary Stabbing Headache	4.1	2
2	Primary Cough Headache	4.2	0
3	Primary Exertional Headache	4.3	0
4	Primary Headache ass with Sexual Activity	4.4	1
5	Primary Hypnic Headache	4.5	1
6	Primary Thunderclap Headache	4.6	0
7	Hemicrania Continuua	4.7	0
8	New Daily Persistent Headache	4.8	4
	TOTAL		8

Epidemiological Data

The patients were analysed for [1] **Age Distribution:**

No	Age Group	No of Patients with Migraine	No of Patients with TTH	No of Patients with TAC
1	0-10yrs	8	0	0
2	11-20yrs	139	7	0
3	21-30yrs	110	19	1
4	31-40yrs	89	25	9
5	41-50yrs	26	40	2
6	51 + yrs	10	9	0

Migraine was most common in the 2nd and 3rd decades, 249 out of 382 patients were between 11-30years of age (11-20yrs - 139 patients, 21-30yrs – 110 patients, 31-40yrs – 89 patients). Majority of the patients (65 out of 100 patients) with Tension type headache were in the 4th and 5th decade of life (21-30yrs - 19 patients, 31-40yrs – 25 patients, 41-50yrs – 40 patients).Trigeminal Autonomic Cephalalgia was more common between 31-40years of age - 9 patients.

In patients with migraine triggered seizures of the total 13, 9 patients were in the age group of 11-20years. Basilar migraine was also found to be common in the age group of 11-20years(7 out of 10) patients and Chronic migraine was noticed in 4th and 5th decade of life predominantly.(35 out of 48)

[2] Gender Distribution: The patients were analysed for gender distribution. Female patients dominated in the category of migraine 262 of the 402 patients, in tension headache 68 of the 100 patients and while in trigeminal autonomic cephalalgia 10 of the 12 patients were male.

No	Sex Distribution	Migraine	TTH	TAC
1	Male	120	32	10
2	Female	262	68	2

A complication of migraine was more common in female patients. Out of the 13 patients with migraine triggered seizures 10 were female and 3 were male. Chronic migraine was predominantly seen in females (32 out of 48 patients).

[3] Family Predilection: Family history was positive in 174 patients (45.54%) with migraine, 22 patients (22%) with tension headache and none with TAC.

No	Family History	Migraine	TTH	TAC
1	Positive F.H	174	22	0
2	Negative. F.H	208	78	12

Clinical Presentations

1. Migraine: Headache in patients with migraine, 286 presented with unilateral headache, of which 193 patients experienced shift of sides while 96 patients had always an unilateral headache. Most of the episodes of migrainous headache in our patients lasted more than 12 to 24 hours [251 patients]. The migrainous headaches were predominantly temporal (217 patients). Most of the patients experienced a throbbing type of headache (263 patients).

No	Duration	Migraine	TTH	TAC
1	1-6 hours	65	19	10
2	7-12 hours	89	25	2
3	13-23 hours	145	28	-
4	More than or equal to 24 hrs	106	28	-

2. Tension Type Headache: In patients with TTH 56 presented with headache of more than 12hours duration, while 25 patients had duration of 6-12 hours and 19 patients had duration of 1-6hours. 62 of these patients who experienced bilateral headaches had headaches confined to the frontal region. Most of the patients experienced aching type of headache (60 patients), while it was band like in 30 patients. In most of these patients the headache radiated to nuchal region and there was associated scalp tenderness during the episodes of headache.

3. Trigeminal Autonomic Cephalalgia: In patients with Trigeminal Autonomic Cephalagia 10 presented with headache of less than 6 hours duration, while 2 patients had duration of 6-12 hours. 2 of the patients experienced bilateral headaches while 10 had unilateral headaches with most (10 patients) confined to the frontal temporal region and was aching in character.

No	Location	Migraine	TTH	TAC
1	Frontal	99	62	0
2	Temporal	217	18	0
3	Occipital	40	16	0
4	Parietal	26	4	2
5	Fronto Temporal	0	0	10

No	Type of Headache	Migraine	TTH	TAC
1	Throbbing	263	7	0
2	Pricking	56	3	1
3	Aching	39	60	11
4	Band Like	0	30	0
5	Diffuse Dull Ache	14	0	0
6	Burning	10	0	0

B] Symptoms, Aggravating and Relieving Factors:

1. Migraine: The predominant premonitary symptom was fatigue (76 patients), followed by, sense of feeling low, irritability, yawning and over

eating in few patients. Associated symptoms in order occurrence being nausea, photophobia, phonophobia, blurring of vision, vomiting and giddiness. Loss of consciousness was reported in 39 patients. Autonomic symptoms like lacrimation, redness and transient syncope were reported in few patients. The most common aggravating factor in our study group was mental stress, while physical stress, head bath, bright sunlight, lack of sleep, travel and consumption of chocolates were also commonly reported. Head bath as an aggravating factor has been observed in 116 patients. The relieving factors were mostly rest and analgesics or topical applications and sleep.

No	Premonitary Symptoms	Migraine	TTH	TAC
1	Yawning	37	1	-
2	Fatigue	76	14	-
3	Irritability	39	19	4
4	Feeling low	64	1	-
5	Over Eating	22	0	-

2. Tension Type Headache: The predominant premonitary symptom was feeling irritability. Associated symptoms being phonophobia (27 patients) or photophobia, nausea or vomiting and in few patients paresthesia and giddiness. The most common aggravating factor in our study group was mental stress, while physical stress, travel, head bath and lack of sleep

were also commonly reported. The relieving factors were mostly rest and analgesics or topical applications, pressure and coffee / tea ingestion.

No	Associated Symptoms	Migraine	TTH	TAC
1	Blurring of Vision	322	0	4
2	Photophobia	365	25	9
3	Phonophobia	338	25	9
4	Nausea	368	19	11
5	Vomiting	158	5	11
6	Giddiness	148	7	0
7	Lacrimation	27	1	9
8	Paresthesia	34	10	0
9	Tremulousness	38	0	0
10	Transient LOC	39	0	0
11	Confusion	19	0	0
12	Redness of Eyes	16	0	11
13	Pain ipsilateral limbs	45	0	0
14	Drooping of eyelids	0	0	7
15	Nasal Stuffiness	0	0	9

3. Trigeminal Autonomic Cephalagia: The predominant associated symptom was nausea, vomiting and redness of eyes (11 patients), with nasal stuffiness, phonophobia, photophobia, drooping of eyelids and blurring of vision being the other common associated symptoms in order of occurrence. Aggravating factors reported this study were alcohol

intake, sunlight and lack of sleep. The relieving factors were mostly analgesics or topical applications, pressure and coffee / tea ingestion.

No	Aggravating Factors	Migraine	TTH	TAC
1	Head Bath	116	15	0
2	Physical Stress	177	22	0
3	Mental Stress	299	25	0
4	Sunlight	212	12	1
5	Travel	187	18	0
6	Lack of Sleep	269	19	2
7	Perfumes	62	0	0
8	Petrol/Diesel Smell	16	0	0
9	Concentrated Reading	32	4	0
10	Chocolates	117	0	0
11	Cold Food/Drink	98	0	0
12	Close Rooms - Theatre/Auditorium	36	8	0
13	Alcohol Intake	0	0	3

No	Releiving Factors	Migraine	TTH	TAC
1	Rest & Analgesics	356	37	0
2	Rest & Topical Applications	126	14	8
3	Sleep	332	11	1
4	Rest	112	6	1
6	Local Pressure	0	26	6
7	Coffee / Tea	18	16	1

C] Aura:

The preponderant type of aura reported in our migranous patients was visual aura in 107 patients while sensory aura was reported in 17 patients and both in 2 patients. The visual aura was predominantly in the form of flickering of lights in 70 patients, while zig-zag lines, scintillating scotomas and fortification spectra was noted in 17 patients, 10 patients and 5 patients respectively. The sensory aura seen was commonly in the form of paresthesia (12 patients).

No	Aura	No of Patients	Type of Aura	
1	Visual Aura	102	Fortification Spectra	5
			Scintillating Scotoma	10
			Flickering Lights	70
			Zig Zag Lines	17
2	Sensory Aura	17	Paresthesia	12
			Numbness	5
3	Visual & Sensory aura	7		

D] Migraine Triggered Seizures:

Of the 13 patients who had Migraine Triggered Seizures 9 patients had Migraine Terminating as Seizures and 4 patients had Migralepsy. On analysing the seizure pattern of these 13 patients, 7 patients had GTCS, 6 patients had CPS.

No	Migraine Triggered Seizures	No of Patients
1	GTCS	7
2	CPS	6

No	Time between Migraine & onset of seizure	No of Patients
1	2-3hrs	3
2	3-6 hrs	6
3	6-12 hrs	3
4	>12 hrs	1

E. Associated Conditions:

1. Migraine: In our study of 382 patients with migraine the following clinical conditions were seen associated – namely Healed granulomatous lesions on CT, Seizures, Hypertension, and Stroke.

2. Tension Type Headache: In our study of 35 patients with TTH the following clinical conditions were seen associated – namely healed granulomatous lesions in CT, Hypertension.

3. Trigeminal Autonomic Cephalagia: In our study of 12 patients with TAC, no significant clinical conditions were associated.

No	Clinical Diagnosis	Migraine	TTH	TAC
1	Seizures	52	0	0
2	Calcified Granuloma on CT	36	16	0
3	Stroke	3	0	0
4	Hypertension	30	15	0

Diagnostic Tests

1. Electrophysiology:

a) Migraine:

EEG was taken in 52 of 382 patients with migraine. EEG was taken in all the 13 patients with Migraine Triggered Seizures. Changes were seen in 8 out of 13 patients, 5 patients had spikes and sharp waves in posterior head region, 3 patients had non specific slowing in posterior regions and there were no specific changes in 5 patients. In other 39 patients, nonspecific slowing in posterior regions was seen in 30 patients and EEG was normal in 9 patients.

(b) Tension Type Headache: EEG was taken in 9 of the 100 patients with TTH, 3 of these patients showed non specific slowing in posterior region, while 6 of the patients had no specific changes

(c) Trigeminal Autonomic Cephalagia: EEG was taken in 2 of the 12 patients with Cluster Headache and none showed specific changes.

No	Migraine -EEG	Migraine	TTH	TAC
1	No Changes	14	6	2
2	Non Specific Slowing in Posterior Region	33	3	0
3	Spikes & Sharp Waves in Occipital Region	5	0	0

2. CT Scan:

(a) Migraine: CT scan of Brain was taken in all of the 382 patients, of whom 40 had changes. The most common change reported in CT scan brain was calcified granulomas in 36 patients, gliosis in 3 patients and basal ganglia calcification in 1patients. In all the 19 patients with Migraine trigerred seizures, CT Scan Brain was found to be normal in all patients.

No	Migraine Neuro Imaging - CT Brain	No of Patients
1	Normal	342
2	Abnormal	40
	Total	382

No	CT Brain In Migraine	No of Patients
1	Calcified Granuloma	36
2	Gliosis	3
3	Basal Ganglia Calcification	1
	TOTAL	40

(b) Tension Type Headache: CT scan of brain was taken in all of the 100 patients, of whom 18 had changes. The most common change reported in CT scan brain was calcified granulomas in 16 patients and gliosis in 2 patients..

No	CT Brain in TTH	No of Patients
1	Normal	82
2	Calcified Granuloma	16
3	Gliosis	2

(c) Trigeminal Autonomic Cephalagia: CT scan of brain was taken in all of the 11 patients with none showing abnormality.

Prophylaxis

1. Migraine: The patients with migraine were given prophylactic therapy with either Propronolol (20-160mg), or Amitryptyline(12.5-50mg), or Propronolol (20-160mg) and Amitryptyline (12.5-50mg) or Sodium Valproate (15 to 20mg/kg).

No	Drugs	Patients
1	Propronolol(20-160mg)	122
2	Amitryptyline(12.5-50mg)	39
3	Propronolol (20-160mg)+ Amitryptyline (12.5-50mg)	218
4	Sodium Valproate (15 to 20 mg / kg)	3
	TOTAL	382

No	Migraine	No of Patients	Disease Free period						Decreased Intensity & Frequency of Pain	%	No Response
			>1 Year	%	6Months-1year	%	Less than 6months	%			
1	Propronolol(20-160mg)	122	15	12	33	27	31	26	43	35	-
2	Amitryptyline(12.5-50mg)	39	4	10	7	18	9	23	14	36	5 (13%)
3	Propronolol (20-160mg)+ Amitryptyline (12.5-50mg)	218	35	16	71	33	62	28	50	23	-
4	Sodium Valproate (15 to 20 mg / kg)	3	-		2	67	-		1	33	-

2. Migraine Triggered Seizures: All patients were treated with anti migrainous prophylaxis with T.Propronolol (40-120mg) and analgesics during episodes of headache. Sodium valproate (200mg thrice a day) was given to patients who had seizures on follow up. Follow up period was 1 year. All patients had 1 year incident free period, while on follow up

3. Tension Type Headaches: All patients in our study were treated with Amitryptiline (10-50mgms). Of the 100 patients 6 patients responded with a disease free interval of 1year, 17 patients with a disease free interval of 6 months to 1 year, 35 patients with a disease free interval of less than 6months, 34 patients responded with decreased intensity and frequency of episodes while 8 patients did not show any response.

4. Trigeminal Autonomic Cephalalgia: One patient with SUNCT responded to prophylaxis with sodium valproate with decreased disease intensity and frequency. Five patients with paroxysmal hemicrania responded well to Indomethacin during acute phases and were given prophylaxis with Amitrptiline. Three patients had a disease free interval of less than six months and two patients had decreased disease intensity and frequency. Prophylaxis was provided for 6 patients with cluster headache of which 4 were given Amitryptiline (25-50mgms), 2 patients were given Propranolol (80-120mgms). Of the 4 patients given

Amitryptiline 3 patients responded with decreased disease intensity and frequency, 1 patient with a disease free interval of 6 months to 1 year. Of the 2 patients given Propranolol, both of them responded with a disease free interval of 1year.

DISCUSSION

This study at the Neurology OPD, Govt. Rajaji Hospital, Madurai between 1st March 2009 and 28th February 2010 encompassed a period of one year.

I DEMOGRAPHY OF PRIMARY HEADACHES

I. INCIDENCE: Of the 502 patients of primary headaches included in this study 382 (76.09%) patients had migraine, 100 (19.92) had tension type headache, 12 (2.39%) had Trigeminal autonomic cephalalgia, 8 (1.57%) had other types of primary headaches. This profile of headaches in our study was in inverse correlation with few international studies. [Lipton et al 2002].[31] Tension type headaches are considered the most common form of headache in the general population with a prevalance of nearly 80% while the prevalance of migraine is pegged at 16% in various international studies[31]. In contrast, migraine is a more common form of headache reported in clinical practice. This variance is attributed to self treatment of tension type headaches by the general population. This variation reported in our study correlates with the study of Lance and Curran [1964] [32]

A. Incidence of Migraine: In our study of the 382 cases of Migraine, 186 (48.69%) patients had Migraine without Aura, 36 (9.53%) patients had Migraine with Aura, 90(23.09%) patients had Migraine which presented with and without aura, 62(17.37%) patients had complications

of migraine and 8(2.09%) patients had probable migraine. The ratio of Migraine without Aura and Migraine with Aura in our study is calculated approximately at 5:1 which correlates with international studies [Allan H Ropper 2005].[33] however the ratio narrows down to 1.5:1 if migraine without aura is compared against migraine with and without aura. In our study of the 382 cases of Migraine, 90 (23.56%) patients had Migraine which presented with or without aura. These 90 patients were placed as a separate group. This group of patients have has been identified and given particular mention in the ICHD 2004 classification. "*Many patients who have frequent attacks with aura also have attacks without aura (code as 1.2 Migraine with aura and 1.1 Migraine without aura).*" [34] Hence in our study we have placed this group of patients as a separate sub entity within the entity migraine

B. Incidence of Tension Type Headaches: In this study of 502 headache patients 100 (19.92%) had tension type headache, of which 16 patients had Infrequent Episodic TTH, 24 patients had Frequent Episodic TTH, 48 patients had Chronic TTH, and 12 patients had Probable TTH. The prevalence of Tension type headache was 40.5% in epidemiological studies by Schwartz et al (1998)[35]. Though tension type headaches are considered the most common form of headache in the general population with a prevalance of nearly 80% in many studies [31], most of the cases are

not reported; many of the patients take self medication, hence the actual number of cases seen by physician is less [32].

C. Incidence of Trigeminal Autonomic Cephalagias: Out of a total of 502 patients registered at the Neurology OPD, 12 (2.39%) had Trigeminal autonomic cephalalgia of which 6 patients had Cluster Headache, 4 patients had Paroxysmal hemicrania, 2 patients had Short Lasting Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing. No case of Probable TAC was registered.

In this study the incidence of cluster headache is 1%, which is in contrast to the observation made by Ambar Chakravarthy et al 2004 that cluster headache appears to be less common in India than in the west [36]. The prevalence of cluster headache estimated in U.S is around 0.1%.

Out of a total of 502 patients registered at the Neurology OPD 8 (1.59%) had other types of primary headaches of which 2 patients had Primary Stabbing Headache, 1 patient had Primary Headache associated with Sexual Activity, 1 patient had Primary Hypnic Headache, and 4 patients had New Daily Persistent Headache

II. AGE AND GENDER DISTRIBUTION OF PRIMARY HEADACHES

A. Age Distribution:

(i) Migraine: Of the 402 patients having migraine most of them were between 11-40years of age (11-20yrs - 139 patients, 21-30yrs – 110 patients, 31-40yrs – 89patients).In this study Migraine with aura peaked around 12-17 years of age in males(5 out of 10) and around 18-22 years in females(9 out of 26). The onset and peaking in both male and female patients in this study is 8-10 years delayed in contrast to that observed in western population by Stewart et al [1991]. According to him the incidence of migraine with aura in females peaked between ages 12 and 13 (14.1/1000 person-years); and in males, migraine with aura peaked in incidence several years earlier, around 5 years of age at 6.6/1000 person-years. Before puberty, migraine prevalence is higher in boys than in girls. The peak incidence of basilar migraine in this study was around 10-15 years which correlates with the international literature. As adolescence approaches, incidence and prevalence increase more rapidly in girls than in boys. The prevalence increases throughout childhood and early adult life until approximately age 40, after which it declines [Stewart WF et al 1991][37] If the migraine headaches persist beyond 40 years of age there is more chance for transformation into chronic migraine.

(ii) Tension Type Headache: Of the 100 patients having Tension type headache most of them were between 31-50years of age (65 out of 100 patients, 31-40yrs – 25 patients, 41-50yrs – 40patients). The average age of onset of TTH is higher than in migraine in this study, namely 25 to 30 years. The prevalence peaks between ages 35 to 45 and decreases slightly with age. This is in consonance with the observation by Rasmussen BK 1995[38].

(iii) Trigeminal Autonomic Cephalagias: Of the 12 patients having cluster headache most of them (9 out of 12 patients) were between 31-40years, which correlates with the observation by Bohra et al.2002[39]

B. Gender Distribution: The patients analysed for gender distribution.

(i) Migraine: Of the 382 patients with migraine 68.58% of them were females. Female predominance is noted in all groups including migraine with aura, migraine without aura, basilar migraine, migraine triggered seizures, migrainous infarction and chronic migraine. Menstrually related migraine was noticed in 24 patients whereas pure menstrual migraine was present in 2 patients. The American Migraine Study-1 (AMS-1)[10] and AMS-II, collected information from 15,000 households representative of the US population in 1989 and 1999. Yet another study, the American Migraine Prevention and Prevalence study (AMPP) replicated the methods of AMS-I and AMS-II. In these three

large studies, the prevalence of migraine was about 18% in women and 6% in men [Abu-Arefeh.I et al 1994][40]

(ii) Tension Type Headache: In tension headache 68 of the 100 patients were female; the female-to-male ratio of TTH in this study is 3:1, again showing a female preponderance. In western countries the ratio is 5:4 indicating that, unlike migraine, women are affected only slightly more than men [Stovner L,et al 2007][41]

(iii) Trigeminal Autonomic Cephalagias: In cluster headache 10 of the 12 patients were male In this study male predominance is noted in concordance with the observation by Manzoni GC. [Manzoni GC et al 1998][42] Of the two female patients included under TAC, one had SUNCT and other had episodic cluster headache**.** .

C. Family Predilection: Family history was positive in 229 patients (45.54%) with migraine. Of the 36 patients with migraine with aura 20 out of 36 patients had a first degree relative suffering from headache.Russell MB et al [1995] has stated that first degree relatives of patients with migraine with aura had a three –four fold increased risk of migraine and it is two fold in first degree relative's patients with migraine with out aura [43]. 22 patients (22%) with tension headache in this study had a positive family history. Family background of some form of headache in 40% of patients with Tension Type Headache has been reported by Freidman A et al [1964][44] while Russell MB et al [1999][45]

reported that parents siblings and children had a 2.1 to 3.9 fold increase risk of chronic TTH during their life time. Our study group does not have a family history in the TAC group even though Kudrow L et al.[1980][46] in his study has reported a high risk of cluster headache by 14 times among first degree relatives of cluster headache patients

III. CLINICAL PRESENTATIONS OF PRIMARY HEADACHES

A) MIGRAINE:

1] Location and Character: In patient with migraine 286 presented with unilateral headache, with 193 of these patients experiencing shift of sides while 96 patients had always a unilateral headache. The headache was predominantly temporal (217 patients). Most of the patients experienced a throbbing type of headache (263 patients) with a majority of cases (145) lasting for a duration of 12-24 hours. The predominant premonitory symptom was fatigue (76patients), with nausea, , photophobia, phonophobia and blurring of vision the commonest associated symptom in order of occurrence. Loss of consciousness was reported in 39 patients.

2] Aura: Among the several types of aura, visual aura was more common (102 of 126 cases) which is in concordance with the literature[47]. Two patients while on treatment for migraine headaches with aura, later had only aura alone without headache. The visual aura was predominantly in the form of flickering of lights in 70 patients, while zig-

zag lines, scintillating scotomas and fortification spectra was noted in 17 patients, 10 patients and 5 patients respectively. This is in correlation with most international studies [Christopher J Boes et al 2004][48]. When patients with typical aura with migraine headache become older, their headache may lose migraine characteristics or disappear completely even though auras continue[47]. Sensory aura was reported in 17 patients and both in 7 patients. The sensory aura seen was commonly in the form of paraesthesia (12 patients).

3] Aggravating and Relieving Factors: In our study the most common aggravating factors were mental stress, while physical stress and lack of sleep were also commonly reported. Head bath as an aggravating factor has been observed in 116 patients. A similar observation has been referred to by Ravishankar et al 2006[18]. This prospective study analysed this unusual trigger link in 94 out of 1000 Indian patients who fulfilled the International Headache Society criteria for migraine. In 11 patients, hair wash was the only trigger; in 45 patients hair wash was one of the triggers and in 38 patients hair wash was a trigger concurrently and in combination with another common trigger. The effect of episodic and long-term prophylaxis in preventing this trigger-like headache has been analysed. The relieving factors were mostly rest and analgesic ingestion and sleep.

4] Associated Clinical Conditions: In our study of 402 patients with migraine the following clinical conditions were seen associated – namely evidence of healed Granuloma, Seizures, Hypertension and Stroke. The risk of stroke may be increased two to threefold in migraine sufferers. Young adult sufferers and women using hormonal contraception appear to be at particular risk. The mechanism of this association though vastly unclear is attributed to chronic abnormalities of cerebral blood vessel tone [Etminan M et al 2005][49]. Women who experience auras have been found to have twice the risk of strokes and heart attacks over non-aura migraine sufferers and women who do not have migraines [Kurth T et al 2006][43]. Similar observation was made in this study and all the patients who had migrainous strokes were female and all of them had migraine with aura. Migraine sufferers seem to be at risk for both thrombotic and hemorrhagic stroke as well as transient ischemic attacks [Becker C et al 2007] [51]

Ten patients had basilar migraine and 7 out of 10 patients were in 1st and 2nd decade. The symptoms experienced by majority of them were vertigo, diplopia, ataxia, parasthesias, hemianopias, and decreased level of consciousness. These observations are in correlation with most international studies. Basilar migraine is common is adolescent age groups. With increasing maturity of the nervous system attacks of

basilar migraine become less common and generally are replaced by migraine without aura [Peatfield RC et al 2000][52].

5] Complication of Migraine:

(i) Chronic Migraine: In our study of the 382 case of Migraine, 62(16-18%) patients had complications of migraine. Of these 62 patients, 48 patients had Chronic Migraine (12 patients of Chronic Migraine since Onset & 36 patients of Episodic Headache converting to Migraine), 1 patient had Migrainous Infarction (Right Occipital Infarction -1) and 13 patients had Migraine Trigerred Seizures (Migraine Terminating as Seizures – 9, Migralepsy - 4). In our study, the most common complication observed in patients with migraine was transformation of migraine to chronic migraine or chronic daily headache. As the chronicity develops migraine headache lost its episodic presentation. Most of these patients with transformed migraine are patients with migraine without aura which is concordant with the studies of Siberstein SD et al 2001[53]. One patient was diagnosed to have migrainous infarction in our study. She was a female of age of 28 years. Women who experience auras have been found to have twice the risk of strokes and heart attacks over non-aura migraine sufferers and women who do not have migraines [Kurth T et al 2006][50, 54].

(ii) Migraine Trigerred Seizures: In our study, we came across 52 instances of Migraine associated with Seizures, 13 patients had

Migraine Triggered Seizures, 17 patients had Post Ictal Migrainous Headache and 22 patients had Migraine & Coexistent Seizure. Of the 13 patients who had Migraine Triggered Seizures, 9 patients had Migraine Terminating as Seizures and 4 patients had Migralepsy. Analysis of the 13 patients showed that 7 patients had GTCS, 6 patients had CPS. Migralepsy as defined in literature are seizures occurring during or within an hour of migrainous aura [Lennox WG et al 1960][55]. In our study of these 13 patients who had migraine which terminated as seizures, 9 patients differed from the classical description of migralepsy in their duration of headache with a window period of more than two to three hours well outside the defined window period of 1 hour. This group of 9 patients presented with both migraine with aura and migraine without aura. All of these 9 patients responded to anti-migrainous prophylaxis alone with a 1year episode free period. Hence these patients were grouped as Migraine triggered seizures. These observations in our study correlate with observations of Anderman F et al (1987)[56]. Marks and Ehernberg et al [1993][57] evaluated and established the relationship between catamenial epilepsy and patients with migraine with aura, showing an increased risk for an association between seizures and migraine. Lenaerts et al [1999][58]evaluated the degree of co-morbidity and established the pattern of temporal relationship between migraine and epilepsy in 202 patients in tertiary care clinics, which outlines that

Migraine attacks equally precede or follow seizures, but migraine aura more often preceded the seizure. In analyzing our group of patients, migraine preceeded the seizures in 13 of 52 patients. Of the 13 patients, 4 patients had migralepsy and 9 patients had migraine which terminated as seizures. Relationship between duration of headache and terminations as seizures was analysed. In our patients, all had a prolonged duration of migraine ranging from 2 to >12hrs duration preceding seizures. This observation in our study is in contrast to the observation of Young et al [1983][59] in their epilepsy unit, where their patients had a brief duration of headache lasting for upto 20 minutes.Headache can also be the sole or most predominant manifestation of epileptic seizures, though this is a relatively rare situation [Laplante P et al 1983][60]

B. TENSION TYPE HEADACHE In patients with TTH 56 presented with headache of more than 12hours duration, while 25 patients had duration of 6-12 hours and 19 patients had a duration of 1-6hours. 62 of these patients experienced bilateral headaches and in most confined to the frontal region. Bendtsen L et al [2006][61] evaluated and compiled the features of tension type headache. TTH is characterized by a bilateral, pressing, tightening pain of mild to moderate intensity, occurring in short episodes of variable duration (episodic forms) or continuously (chronic form). The headache is not associated with the typical migraine features, such as vomiting, severe photophobia, and phonophobia. In the chronic

form, only one of these accompanying symptoms is allowed and only mild nausea is accepted. Because of lack of accompanying symptoms and milder pain intensity, patients rarely are severely incapacitated by their pain. TTH is the most featureless of the primary headaches and, because many secondary headaches may mimic TTH, a diagnosis of TTH requires exclusion of other organic disorders. A general and neurologic examination and prospective follow-up using headache diaries with registration of all consumed drugs are, therefore of utmost importance to reach a diagnosis. There are no reliable specific paraclinical tests that are useful in differential diagnosis. Manual palpation of the pericranial muscles and their insertions should be done to demonstrate a possible muscular factor for patients and to plan treatment strategy, where physical training and relaxation therapy are important components. [Jensen R 1999][62]

Most of the patients experienced aching type of headache (60 patients), while it was band like in 30 patients and throbbing in 7 patients. The predominant premonitary symptom was irritability (19 patients) with photophobia, phonophobia, nausea were other common associated symptoms in order of occurrence. The most common aggravating factor in our study group was mental stress, while physical stress, travel, head bath and lack of sleep were also commonly reported. The relieving factors were mostly pressure and analgesics and coffee / tea ingestion. As

TTH was commonly associated with depression and is aggravated by mental stress, previously was thought to be psychogenic now a neurological basis was established. Other common aggravating factors include poor self related health, inability to relax after work and sleeping few hours per night (Bendsten L etal 2000)[63]. In our study of 100 patients with TTH the following clinical conditions were seen associated – namely evidence of healed Granuloma, Hypertension.

C. CLUSTER HEADACHE & OTHER TRIGEMINAL AUTONOMIC CEPHALAGIAS

In patients with TAC, 10 presented with headache of less than 6 hours duration, while 2 patients had duration of 6-12 hours. One of the patients experienced bilateral headaches while 11 had unilateral headaches with most (10 patients) confined to the frontal temporal region. Most of the patients experienced an aching type of headache (11 patients), while it was pricking in 1 patient. The predominant premonitary symptom was nausea and vomiting and redness of eyes with nasal stuffiness (11 patients), phonophobia, photophobia, drooping of eyelids and blurring of vision being the other common associated symptoms in order of occurrence. Aggravating factors that were reported in most patients in our study were alcohol intake, sunlight and lack of sleep. The relieving factors were mostly analgesics, pressure and coffee / tea ingestion. Our observations are in consonance with Rozen TD et al [1999][64] describing

the features of typical cluster headache. Typical cluster headache location is retro-orbital, periorbital, and occipitonuchal. Maximum pain is normally retro-orbital in more than 70% of patients. Pain quality is described as boring, stabbing, burning, or squeezing. Cluster headache intensity is always severe and never mild, although headache pain intensity may be less at the beginning and end of cluster periods. The duration of individual cluster headaches is between 15 minutes and 180 minutes, with more than 75% attacks lasting less than 60 minutes. Attack frequency is between one and three attacks per day, with most patients experiencing two or fewer headaches in a day. Cluster headache is marked by its associated autonomic symptoms that typically occur on the same side as the head pain but can be bilateral. Lacrimation is the most common associated symptom, occurring in 73% of patients, followed by conjunctival injection in 60%, nasal congestion in 42%, rhinorrhea in 22%, and a partial Horner's syndrome in 16% to 84%.

IV DIAGNOSTIC STUDIES

A. Electrophysiology:

(i) Migraine: EEG was taken in 52 of the 382 patients with migraine. 33 of the 52 patients showed non specific slowing in Posterior Region, while 5 patients showed Spikes & Sharp Waves in Occipital Region while 14 of the patients had no specific changes. The EEG

features in our patients were sharp waves and spikes in posterior occipital region mainly occipital region (5 patients of migraine triggered seizures), more during the period of aura and was normal during the interval period between attacks of migraine. This correlates with a large multicenter study - incidence of spikes and paroxysmal events was 12.5% compared to 0.7% in normal adult volunteers. The percentage goes still high up in patients with seizures. Monomorphic or polymorphic slow waves, seen bilaterally. Few cases also showed slowing in posterior head region [Beaumanoir A et al 1987][65].

(ii) TTH: EEG was taken in 9 of the 100 patients with TTH, 3 of these patients showed non specific slowing in Posterior Region, while 6 of the patients had no specific changes.

(iii) TAC: EEG was taken in 2 of the 12 patients with Cluster Headache, these 2 patients showed no specific changes.

B. Radio-Imaging Studies: (i) Migraine: CT scan of brain was taken in all of the 382 patients, of whom 40 had changes. The most common change reported in CT scan brain was calcified granulomas in 36 patients, gliosis in 3 patients and basal ganglia calcification in 1 patient. Frishberg BM et al 1994[66] reviewed four CT scan studies, four MRI scan studies, and one combined MRI and CT scan study of 897 scans of patients who had migraine. These findings are combined with more recent reports of one CT scan study of 284 patients and six studies

of MRI scans of 444 patients for a total of 1625 scans of patients who had various types of migraine. Other than white matter abnormalities, the studies showed no significant pathology except for four brain tumours (three of which were incidental findings) and one AVM (in a patient who had migraine and a seizure disorder).

V. PROPHYLAXIS

A. Migraine: The patients with migraine were given prophylactic therapy with either Propronolol (20-160mg) or Amitryptyline (12.5-50mg) or Propronolol (20-160mg) and Amitryptyline (12.5-50mg) or Sodium Valproate ER(15 to 20mg/kg). Patients with all types of migraine responded well to prophylactic therapy with headache free interval of more than six months in majority. Among 218 patients with migraine who were on combination prophylaxis with Amitryptiline and Propronolol 35 (16%) responded with disease free period of more than 1 year and a further 71(33%) responded with disease free period of more than 6 months, 62(28%) patients responded with disease free period of less than 6 months while the remaining 50(23%) responded with decreased intensity and frequency of pain. Among 122 patients who were on Propronolol alone 15 (12%) responded with disease free period of more than 1 year, 33(27%) responded with disease free period of more than 6 months, 31(26%) patients responded with disease free period of less than 6 months while the remaining 43(35%) responded with

decreased intensity and frequency of pain. Among the 39 patients on Amitryptiline 4 (10%) responded with disease free period of more than 1 year, 7(18%) responded with disease free period of more than 6 months,9 (23%) patients responded with disease free period of less than 6 months, 14(36%) responded with decreased intensity and frequency of pain and the remaining 5 (13%) showed no response. Among the 3 patients treated with sodium valproate 2(67%) responded with disease free period of more than 6 months and 1(33%) patient responded with decreased intensity and frequency of pain.

This is in correlation with meta analysis from many studies which showed that beta blockers were associated with reduction in migraine activity and more than 100 clinical trials clearly established the benefits of propronolol [Linde K et all] [67]. In a systematic review of 26 clinical trials, propranolol was shown to be more effective than placebo in reducing migraine frequency among adults. Disease free interwal of 1 year period for patients on migraine prophylaxis varied from 8 to 22% [Linde et all 2004].[67] Propronolol significantly reduced the headache index(a composite score that takes into account both intensity and duration) in 56% of patients[68] [Modi S et all] In a double-blind controlled clinical trial Amitriptyline reduced both intensity and duration of migraine attacks in 44% of cases[Gomersall and Stuart][69]. In a double-blind study of sodium valproate versus placebo Hering R et all[70]

found effectiveness of valproate in reducing severity and frequency of migraine attacks in 86.2% of patients.

B. Tension Type Headaches: All patients in our study were treated with Amitryptiline (10-50mgms). Of the 100 patients 6 patients responded with a disease free interval of 1year, 17 patients with a disease free interval of 6 months to 1 year, 35 patients with a disease free interval of less than 6months, 34 patients responded with decreased intensity and frequency of episodes while 8 patients did not show any response. This is in concordance with various studies. Prophylactic pharmacotherapy include tricyclic antidepressant, amitriptyline is the only drug proved effective in several controlled trials in TTH. [Bendtsen L et al 2005][71]. The two most recent studies reported that amitriptyline (75 mg per day) reduced headache duration and intensity by 30% compared with placebo. The effect is long lasting (at least 6 months) and not related to the presence of depression. [Bendtsen L et al 1996][72]. If patients do not respond to amitriptyline, mirtazapine could be attempted. Venlafaxine or SSRIs could be considered in patients who have concomitant depression, if tricyclics or mirtazapine are not tolerated.

C. Trigeminal Autonomic Cephalalgia: One patient with SUNCT responded to prophylaxis with sodium valproate with decreased disease intensity and frequency. Five patients with paroxysmal

hemicrania responded well to Indomethacin during acute phases and were given prophylaxis with Amitrptiline with good response. Three patients had a disease free interval of less than six months and two patients had decreased disease intensity and frequency. Prophylaxis was provided for 6 patients with cluster headache of which 4 were given Amitryptiline (25-50mgms), 2 patients were given Propranolol (80-120mgms). Of the 4 patients given Amitryptiline 3 patients responded with decreased disease intensity and frequency, 1 patient with a disease free interval of 6 months to 1 year. Of the 2 patients given Propranolol, both of them responded with a disease free interval of 1year. These drug responses are at variance to usual observations, these patients showing marked responses indicating that these were probable Cluster Headache Migraine overlap syndromes. Solomon S et al [1986][73] has referred to a similar clinical situation were the patients had clinical features of both types of headaches. Graham et al [1975][74] described combined syndromes and has described migraine headaches in recurrent bouts resembling cluster headache responding to migraine prophylaxis.

CONCLUSIONS

The observations of this study are here with summarised

1. Migraine is the commonest type of headache comprising of about 76%. Migraine without aura [48%] was more common than migraine with aura [32%]. Female preponderance was noticed in all subtypes of migraine, age of onset being in 2nd and 3rd decade for majority of the subgroups of migraine, except for basilar migraine which was common in 1st and 2nd decade. Migraine pain was temporal in location, unilateral, throbbing in character, lasting for 12 to 24 hours in majority of the cases.
2. Chronic migraine, Migraine triggered seizures and Migrainous infarction were the complications of migraine encountered in this study in the order of frequency of occurrence. Transformation to Chronic migraine was more common from episodic forms and in patients with onset of migraine in teens or twenties. Among patients with migraine and seizures, 13 patients were grouped under migraine triggered seizures that differed from the classical description of migralepsy in their duration of headache, with a window period of more than two hours prior to the seizure. This

group of patients presented with both migraine with aura and migraine without aura and responded to anti-migrainous prophylaxis alone with a 1 year episode free period.

3. Tension type headache constituted about 20% of cases, Chronic tension type headache was the commonest type (48%), majority of cases occurred in fourth decade with female preponderance.
4. Patients with all types of migraine responded well to Antimigraine prophylactic drugs. Among migraine patients on combination prophylaxis with Propronolol and amitryptiline, 16% responded with disease free interwal of more than 1 year, while it was 12% for patients on propronolol and 10% for patients on amitryptiline. The response rate with disease free interwal in the range of 6 to 12 months was 33%, 27% and 18% for the above respective group of drugs.

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APPENDIX-1

PROFORMA FOR HEADACHE

Name: **Age:** **Sex:**

Residential address:

Occupation:

NM No:

Details about headache: How long-

Age of onset-

Headache free period if any-

Duration in hours-

Location- Unilateral- Bilateral-

Temporal- Frontal - Occipital - Holocranial-

Radiation to neck- Retro orbital region- Limbs (UL & LL) –

Character- Throbbing- Tight sensation- Shock like-

Pricking- Aching-

Frequency- Episodes/week- Episodes/month-

Recent change in frequency-

Frequency with prophylactic drugs-

Associated features- Photophobia and phonophobia-

Nausea and vomiting-

Drowsiness-

Giddiness/ vertigo-

LOC-

Redness of eyes-

Lacrimation-

Diplopia-

Parasthesias-

Aura- Visual- phenomena:

Positive visual phenomena: Small bright dots-

White spots/ flashes of light (photopsias)-

Fortification spectra (teichopsia)-

Other zig zag lines-

Coloured spots of light-

Negative visual phenomena: Blind spots (scotomas)-

Black dots/ spots-

Hemianopias-

Disturbances of visual perception- blurred vision-

Micropsia/ macropsia/teleopsia-

Tunnel vision-

Triggering factors- Physical stress/ mental stress-

Sunlight-

Sleep alteration-

Travel-

Headbath-

Perfumes/fumes-

Cold items-

Position of neck-

Others-

Relieving factors- Pressure-

Coffee/ tea-

Analgesics

Rest-.

Relationship to menstrual cycles-

Family history-

On Examination- BP-

Temporal artery-

Sinuses-

Palpabrel fissure-

Refraction-

Fundii-

Cranial nerves-

Long tract signs-

Associated conditions- Seizures-

HT-

Stroke-

Granulomatous diseases-

Psychiatric diseases-

Investigation- CT scan Brain-

EEG-

MRI Brain-

Diagnosis- **Primary/ Secondary-**

Migraine / Tension type headache / Short lasting headache /

Cluster headache / Paroxysmal hemicranias

Medications- Propranolol

Sodium valproate

Amitryptyline

Flunarizine

Paracetamol

Other NSAIDs

Follow up-

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
1	35	F	18	Temporal	Throbbing	6	Visual				MWA	prop	DFI-12M	MWA – Migraine with Aura
2	36	F	6	TEMPORAL	ACHE	4					PTTH	Amitryptiline	DIF	MWOA – Migraine without Aura
3	31	F	14	Temporal	Throbbing	20					CM	amt + prop	DIF	MWWOA - Migraine with and without Aura
4	38	F	20	OCCIPITAL	PRICKING	3					IETTH	Amitryptiline	DFI-6M	CM- Complications of Migraine
5	17	M	15	Temporal	Pricking	4	Sensory & Visual				MWA	prop	DIF	PM- Probable Migraine
6	33	F	30	Temporal	Throbbing	3					MWOA	prop	DIF	TTH-Tension type Head ache
7	43	F	7	OCCIPITAL	ACHE	11					FETTH	Amitryptiline	DIF	FETTH-Frequent Episodic Tension type Head ache
8	39	M	10	Frontotemp	Aching	15					CCH	propronolol	DFI-12M	IETTH-Infrequent Episodic Tension type head ache
9	8	F	18	Occipital	Aching	12					MWOA	prop	DFI-6M	PTTH- Probable Tension type Head ache
10	50	F	24	Frontal	Throbbing	16					CM	amt + prop	DIF	CTTH-Chronic Tension type head ache
11	37	M	18	Temporal	Throbbing	6	Sensory				MWWOA	amt + prop	DIF	TAC- Trigeminal Autonomic Cephalagias
12	32	M	14	Temporal	Burning	5					MWOA	prop	DIF	CCH – Chronic Cluster Headache
13	18	F	18	Parietal	Throbbing	8					MWOA	prop	DIF	ECH –Episodic Cluster Headache
14	15	M	18	Temporal	Pricking	2					MWOA	prop	DIF	EPHC – Eposodic Paroxysmal Hemicrania
15	32	F	12	Frontal	Throbbing	12					PM	amt + prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
16	15	F	24	Frontal	Throbbing	15					CM	amt + prop	DIF	DFI-12m- Disease free interval for 12 months
17	29	M	3	OCCIPITAL	ACHE	20					CTTH	Amitryptiline	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
18	32	F	9	Parietal	Diffuse	6					MWOA	amt + prop	DIF	DFI-6m - Disease free interval less than 6 months
19	44	F	14	Frontal	Throbbing	20					CM	amt + prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
20	33	M	20	Temporal	Throbbing	8	Visual				MWWOA	amt + prop	DIF	MWA – Migraine with Aura
21	35	F	12	Parietal	Throbbing	6	Visual				MWA	prop	DIF	MWOA – Migraine without Aura
22	36	M	15	Temporal	Throbbing	6					PM	amt + prop	DIF	MWWOA - Migraine with and without Aura
23	17	F	24	OCCIPITAL	ACHE	7					FETTH	Amitryptiline	DIF	CM- Complications of Migraine
24	14	F	18	Temporal	Aching	4					MWOA	prop	DIF	PM- Probable Migraine
25	38	F	15	Temporal	Aching	2	Sensory				MWWOA	amt + prop	DIF	TTH-Tension type Head ache
26	10	F	18	Temporal	Throbbing	6					MWOA	prop	DFI-12M	FETTH-Frequent Episodic Tension type Head ache
27	42	F	22	OCCIPITAL	ACHE	15					CTTH	Amitryptiline	DIF	IETTH-Infrequent Episodic Tension type head ache
28	42	F	12	Frontal	Throbbing	18					CM	amt + prop	DFI-6M+	PTTH- Probable Tension type Head ache
29	33	F	14	Temporal	Throbbing	8	Visual				MWWOA	amt + prop	DIF	CTTH-Chronic Tension type head ache
30	38	M	18	Temporal	Pricking	2					MWOA	prop	DIF	TAC- Trigeminal Autonomic Cephalagias
31	16	F	3	TEMPORAL	THROBBING	4					IETTH	Amitryptiline	DFI-6M+	CCH – Chronic Cluster Headache
32	33	M	6	Frontotemp	Aching	24					ECH	Amitryptiline	DFI-6M+	ECH –Episodic Cluster Headache
33	33	M	8	Temporal	Pricking	4					MWOA	prop	DFI-6M	EPHC – Eposodic Paroxysmal Hemicrania
34	31	M	24	Temporal	Throbbing	6	Visual				MWWOA	amt + prop	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
35	28	F	24	OCCIPITAL	ACHE	5					PTTH	Amitryptiline	DIF	DFI-12m- Disease free interval for 12 months
36	15	F	12	Temporal	Aching	6					MWOA	prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
37	32	F	18	Frontal	Throbbing	2	Visual				MWWOA	amt + prop	DIF	DFI-6m - Disease free interval less than 6 months
38	29	F	3	OCCIPITAL	BAND	3					IETTH	Amitryptiline	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
39	40	F	18	Frontal	Pricking	3					MWOA	prop	DIF	MWA – Migraine with Aura
40	32	F	24	Temporal	Throbbing	4	Visual				MWA	prop	DFI-12M	MWOA – Migraine without Aura
41	38	F	18	TEMPORAL	ACHE	20					CTTH	Amitryptiline	DFI-6M+	MWWOA - Migraine with and without Aura
42	35	F	18	Parietal	Throbbing	5					MWOA	prop	DIF	CM- Complications of Migraine
43	48	M	16	FRONTAL	PRICKING	10					FETTH	Amitryptiline	DFI-6M	PM- Probable Migraine
44	20	F	18	Temporal	Throbbing	8	Visual				MWA	amt + prop	DFI-6M+	TTH-Tension type Head ache
45	37	M	24	Temporal	Pricking	10					MWOA	amt + prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
46	31	F	30	Frontal	Aching	12					MWOA	amt + prop	DIF	IETTH-Infrequent Episodic Tension type head ache
47	32	F	7	Frontal	Throbbing	4	Visual				MWA	prop	DFI-12M	PTTH- Probable Tension type Head ache
48	42	F	14	Temporal	Throbbing	2					MWOA	prop	DIF	CTTH-Chronic Tension type head ache
49	45	F	15	Temporal	Aching	2	Sensory				MWWOA	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
50	17	M	24	Temporal	Pricking	3	Visual				MWWOA	prop	DIF	CCH – Chronic Cluster Headache
51	36	F	15	Frontal	Throbbing	22					CM	amt + prop	DFI-6M+	ECH –Episodic Cluster Headache
52	14	F	9	Temporal	Pricking	4	sensory				MWWOA	amt + prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
53	47	M	24	Temporal	Diffuse	8					MWOA	prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
54	35	F	14	Temporal	Throbbing	5					MWOA	prop	DIF	DFI-12m- Disease free interval for 12 months
55	12	M	18	Temporal	Throbbing	4	Sensory & Visual				MWA	prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
56	18	M	24	Frontal	Diffuse	11					MWOA	prop	DIF	DFI-6m - Disease free interval less than 6 months
57	36	F	15	Temporal	Throbbing	12					MWOA	prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
58	13	M	18	Occipital	Throbbing	3	Visual				MWA	prop	DFI-12M	MWA – Migraine with Aura
59	49	F	18	Temporal	Throbbing	6	Visual		gliosis	Stroke	MWA	amt + prop	DFI-6M+	MWOA – Migraine without Aura
60	14	M	14	Frontal	Throbbing	7					MWOA	amt + prop	DIF	MWWOA - Migraine with and without Aura
61	12	M	18	Temporal	Throbbing	2	sensory				MWWOA	amt + prop	DFI-6M+	CM- Complications of Migraine
62	48	M	16	FRONTAL	ACHE	10					FETTH	Amitryptiline	DFI-6M	PM- Probable Migraine
63	15	F	24	Frontal	Throbbing	20					CM	amt + prop	DFI-12M	TTH-Tension type Head ache
64	16	F	18	Parietal	Pricking	5					MWOA	prop	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
65	40	F	12	Temporal	Throbbing	19					CM	amt + prop	DIF	IETTH-Infrequent Episodic Tension type head ache
66	38	M	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	PTTH- Probable Tension type Head ache
67	25	F	15	Temporal	Aching	7					MWOA	prop	DFI-6M+	CTTH-Chronic Tension type head ache
68	18	M	13	Frontal	Throbbing	5	Visual				MWWOA	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
69	36	F	5MIN	Frontotemp	Aching	30					ECH	Amitryptiline	DIF	CCH – Chronic Cluster Headache
70	22	M	24	Frontal	Throbbing	4					MWOA	prop	DFI-6M+	ECH –Episodic Cluster Headache
71	38	F	18	Temporal	Aching	10					MWOA	prop	DIF	EPHC – Eposodic Paroxysmal Hemicrania
72	19	F	18	TEMPORAL	ACHE	20					CTTH	Amitryptiline	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
73	12	M	14	Frontal	Throbbing	8					MWOA	prop	DIF	DFI-12m- Disease free interval for 12 months
74	40	F	24	Temporal	Throbbing	3	Visual				MWWOA	amt + prop	DFI-6M+	DFI-6m+ - Disease free interval 6months -12 months
75	34	M	4	TEMPORAL	ACHE	16					CTTH	Amitryptiline	DFI-12M	DFI-6m - Disease free interval less than 6 months
76	24	F	18	Temporal	Pricking	4					MWOA	amt + prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
77	18	F	14	Frontal	Throbbing	2	Visual				MWA	prop	DFI-6M+	MWA – Migraine with Aura
78	17	F	4	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	MWOA – Migraine without Aura
79	45	F	24	Temporal	Throbbing	19					CM	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
80	35	F	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	CM- Complications of Migraine
81	16	F	18	Parietal	Aching	5					MWOA	prop	DIF	PM- Probable Migraine
82	15	F	18	Temporal	Throbbing	8	Visual				MWWOA	amt + prop	DFI-6M+	TTH-Tension type Head ache
83	28	F	14	Temporal	Throbbing	11					MWOA	prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
84	36	F	14	OCCIPITAL	ACHE	3					PTTH	Amitryptiline	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
85	14	M	7	Frontal	Aching	2				LOC	MWOA	amt + prop	DIF	PTTH- Probable Tension type Head ache
86	22	F	15	FRONTAL	ACHE	12					IETTH	Amitryptiline	DIF	CTTH-Chronic Tension type head ache
87	38	M	24	Temporal	Aching	20					CM	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
88	53	F	18	Temporal	Throbbing	18					CM	amt + prop	DIF	CCH – Chronic Cluster Headache
89	36	F	6	FRONTAL	ACHE	4		YES			PTTH	Amitryptiline	DIF	ECH –Episodic Cluster Headache
90	26	M	18	Frontal	Throbbing	3					MWOA	Amit	NR	EPHC – Eposodic Paroxysmal Hemicrania
91	45	M	36	FRONTAL	ACHE	2			Gliosis		IETTH	Amitryptiline	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
92	24	F	18	Frontal	Throbbing	8					MWOA	prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
93	32	F	24	Temporal	Pricking	2					MWOA	Amit	DIF	DFI-6m+ - Disease free interval 6months -12 months
94	36	F	15	Frontal	Throbbing	22					CM	amt + prop	DFI-6M+	DFI-6m - Disease free interval less than 6 months
95	49	F	9	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
96	42	F	9	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	MWA – Migraine with Aura
97	39	M	6	Frontotemp	Aching	30					CCH	propronolol	DFI-12M	MWOA – Migraine without Aura
98	18	F	14	Temporal	Throbbing	4	Visual				MWA	prop	DFI-6M+	MWWOA - Migraine with and without Aura
99	29	F	8	OCCIPITAL	BAND	3					IETTH	Amitryptiline	DIF	CM- Complications of Migraine
100	36	M	24	Temporal	Throbbing	15					CM	amt + prop	DFI-6M+	PM- Probable Migraine
101	14	M	24	Temporal	Throbbing	3					MWOA	prop	DFI-6M	TTH-Tension type Head ache
102	17	F	4	Parietal	Burning	5					MWOA	amt + prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
103	18	F	16	Frontal	Throbbing	4					MWOA	prop	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
104	18	M	13	Frontal	Throbbing	5	Visual				MWWOA	amt + prop	DFI-6M+	PTTH- Probable Tension type Head ache
105	9	M	4	Occipital	Aching	2	Visual				MWA	prop	DFI-6M	CTTH-Chronic Tension type head ache
106	43	F	7	FRONTAL	BAND	11			calcific granuloma		FETTH	Amitryptiline	DIF	TAC- Trigeminal Autonomic Cephalagias
107	42	F	14	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+	CCH – Chronic Cluster Headache
108	26	F	12	Temporal	Throbbing	2	Visual				MWA	prop	DFI-6M	ECH –Episodic Cluster Headache
109	34	M	24	Temporal	Pricking	4					MWOA	Amit	DIF	EPHC – Eposodic Paroxysmal Hemicrania
110	43	F	9	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
111	24	F	18	Frontal	Throbbing	8					MWOA	prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
112	22	M	15	Temporal	Throbbing	6					PM	amt + prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
113	32	F	12	Temporal	Throbbing	5					MWOA	prop	DFI-6M+	DFI-6m - Disease free interval less than 6 months
114	47	F	9	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
115	14	F	18	Occipital	Aching	3	Visual				MWWOA	prop	DFI-12M	MWA – Migraine with Aura
116	34	M	8	Frontotemp	Aching	60					EEH	Amitryptiline	DIF	MWOA – Migraine without Aura
117	45	F	24	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
118	28	M	18	Temporal	Pricking	8	Visual				MWWOA	amt + prop	DIF	CM- Complications of Migraine
119	24	M	12	Frontal	Throbbing	6					MWOA	amt + prop	DFI-6M+	PM- Probable Migraine
120	26	F	24	Temporal	Throbbing	4	Visual				MWWOA	prop	DIF	TTH-Tension type Head ache
121	15	F	18	Temporal	Throbbing	2					MWOA	prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
122	13	F	18	Occipital	Throbbing	5	Visual				MWWOA	amt + prop	DIF	IETTH-Infrequent Episodic Tension type head ache
123	45	F	36	TEMPORAL	BAND	9					IETTH	Amitryptiline	NR	PTTH- Probable Tension type Head ache
124	49	M	6	Frontotemp	Aching	30					EEH	Amitryptiline	DIF	CTTH-Chronic Tension type head ache
125	53	F	24	Temporal	Diffuse	18					CM	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
126	18	F	14	Temporal	Throbbing	3					MWOA	amt + prop	DFI-6M+	CCH – Chronic Cluster Headache
127	38	M	12	Frontal	Throbbing	5					MWOA	Amit	DFI-6M	ECH –Episodic Cluster Headache
128	22	F	18	Temporal	Throbbing	2	Visual				MWA	prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
129	20	F	18	Occipital	Aching	1					MWOA	prop	DFI-12M	CPHC – Chronic Paroxysmal Hemicrania
130	48	F	5	FRONTAL	THROBBING	17					CTTH	Amitryptiline	DFI-6M	DFI-12m- Disease free interval for 12 months
131	28	M	6	Temporal	Throbbing	8					MWOA	amt + prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
132	22	M	15	Temporal	Throbbing	6					PM	amt + prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
133	36	F	18	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
134	34	M	18	Frontal	Throbbing	7	Visual				MWWOA	Amit	DFI-6M+	MWA – Migraine with Aura
135	45	M	24	FRONTAL	ACHE	2			Gliosis		IETTH	Amitryptiline	DFI-6M	MWOA – Migraine without Aura
136	16	F	14	Frontal	Throbbing	4					MWOA	prop	DFI-6M+	MWWOA - Migraine with and without Aura
137	18	F	24	Temporal	Pricking	8	Visual				MWWOA	amt + prop	DFI-6M	CM- Complications of Migraine
138	45	F	36	TEMPORAL	BAND	9					IETTH	Amitryptiline	NR	PM- Probable Migraine
139	20	F	8	Temporal	Pricking	3	Visual				MWWOA	amt + prop	DFI-6M+	TTH-Tension type Head ache
140	19	F	18	Frontal	Throbbing	2					MWOA	Amit	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
141	48	M	16	FRONTAL	ACHE	10		YES			FETTH	Amitryptiline	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
142	18	F	5	Temporal	Throbbing	4					MWOA	amt + prop	DFI-6M+	PTTH- Probable Tension type Head ache
143	27	M	20	FRONTAL	ACHE	20					CTTH	Amitryptiline	DFI-6M	CTTH-Chronic Tension type head ache
144	24	M	24	Temporal	Throbbing	2	Visual				MWA	prop	DFI-6M	TAC- Trigeminal Autonomic Cephalagias
145	28	F	24	FRONTAL	BAND	5					PTTH	Amitryptiline	DIF	CCH – Chronic Cluster Headache
146	45	F	24	Temporal	Throbbing	20					CM	amt + prop	DFI-6M+	ECH –Episodic Cluster Headache
147	16	F	12	Temporal	Throbbing	5					MWOA	amt + prop	DFI-6M	EPHC – Eposodic Paroxysmal Hemicrania
148	42	F	18	Frontal	Throbbing	15					MWOA	amt + prop	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
149	36	M	24	Temporal	Pricking	3					MWOA	amt + prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
150	53	F	5	OCCIPITAL	ACHE	15		YES			CTTH	Amitryptiline	DIF	DFI-6m+ - Disease free interval 6months -12 months
151	15	F	18	Temporal	Throbbing	5					CM	prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
152	37	M	3	Frontotemp	Aching	30					EPHC	Amitryptiline	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
153	24	F	18	Occipital	Throbbing	2	Visual				MWWOA	amt + prop	DFI-6M+	MWA – Migraine with Aura
154	48	M	16	FRONTAL	ACHE	10		YES	calcific granuloma		FETTH	Amitryptiline	DFI-6M	MWOA – Migraine without Aura
155	28	F	18	Temporal	Throbbing	4		Yes			MWOA	Amit	DFI-6M+	MWWOA - Migraine with and without Aura
156	32	M	6	Temporal	Throbbing	9					MWOA	prop	DFI-6M	CM- Complications of Migraine
157	19	F	24	Frontal	Throbbing	12	Visual				MWWOA	amt + prop	DFI-6M+	PM- Probable Migraine
158	14	F	24	Temporal	Pricking	4					MWOA	amt + prop	DFI-6M+	TTH-Tension type Head ache
159	12	M	18	Frontal	Throbbing	4					MWOA	amt + prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
160	25	F	6	Temporal	Throbbing	6	Sensory & Visual				MWWOA	prop	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
161	18	F	12	Temporal	Throbbing	5	visual				MWA	Amit	DFI-12M	PTTH- Probable Tension type Head ache
162	38	F	18	TEMPORAL	ACHE	20		YES			CTTH	Amitryptiline	DFI-6M+	CTTH-Chronic Tension type head ache
163	20	M	18	Temporal	Pricking	4					MWOA	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
164	22	F	14	Parietal	Throbbing	2	Visual				MWA	amt + prop	DFI-6M	CCH – Chronic Cluster Headache
165	44	F	24	Temporal	Throbbing	5					MWOA	prop	DFI-6M+	ECH –Episodic Cluster Headache
166	18	F	9	Frontal	Throbbing	20					CM	amt + prop	DFI-6M	EPHC – Eposodic Paroxysmal Hemicrania
167	16	M	5	Temporal	Throbbing	3	Visual				MWWOA	amt + prop	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
168	22	M	18	Temporal	Throbbing	5	Visual				MWWOA	amt + prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
169	43	F	7	FRONTAL	BAND	11					FETTH	Amitryptiline	DIF	DFI-6m+ - Disease free interval 6months -12 months
170	24	M	18	Temporal	Throbbing	8					MWOA	Amit	DFI-6M	DFI-6m - Disease free interval less than 6 months
171	8	F	24	Temporal	Throbbing	12					MWOA	amt + prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
172	28	F	24	FRONTAL	BAND	5					PTTH	Amitryptiline	DIF	MWA – Migraine with Aura
173	29	F	4	Occipital	Aching	7	Visual				MWA	prop	DFI-6M	MWOA – Migraine without Aura
174	19	F	24	Temporal	Throbbing	4					MWOA	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
175	17	F	18	Temporal	Throbbing	8					MWOA	amt + prop	DFI-6M+	CM- Complications of Migraine
176	25	F	12	Temporal	Pricking	3	Visual				MWWOA	amt + prop	DIF	PM- Probable Migraine
177	37	F	10	FRONTAL	BAND	10					FETTH	Amitryptiline	NR	TTH-Tension type Head ache
178	33	F	24	Parietal	Throbbing	2					MWOA	amt + prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
179	39	M	24	Temporal	Throbbing	4					MWOA	prop	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
180	43	F	18	Temporal	Throbbing	2					MWOA	amt + prop	DIF	PTTH- Probable Tension type Head ache
181	18	F	24	Temporal	Throbbing	20					MWOA	amt + prop	DFI-6M+	CTTH-Chronic Tension type head ache
182	46	M	18	Temporal	Throbbing	5			gliosis	Stroke	CM	amt + prop	DIF	TAC- Trigeminal Autonomic Cephalagias
183	26	F	18	Occipital	Aching	4	Visual				MWWOA	amt + prop	DFI-6M+	CCH – Chronic Cluster Headache
184	23	F	3	Temporal	Burning	8					MWOA	prop	DFI-6M	ECH –Episodic Cluster Headache
185	31	F	6	Frontotemp	Aching	30					EPHC	Amitryptiline	DFI-6M	EPHC – Eposodic Paroxysmal Hemicrania
186	40	F	12	Frontal	Throbbing	5					MWOA	prop	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
187	22	M	24	Temporal	Throbbing	3	Visual				MWA	amt + prop	DIF	DFI-12m- Disease free interval for 12 months
188	14	M	18	Temporal	Pricking	12	Visual				MWA	prop	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
189	18	F	2	Temporal	Throbbing	7					MWOA	amt + prop	DFI-6M+	DFI-6m - Disease free interval less than 6 months
190	32	F	18	Temporal	Throbbing	14	Sensory				MWWOA	amt + prop	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
191	20	F	18	Frontal	Throbbing	12					MWOA	prop	DFI-6M+	MWA – Migraine with Aura
192	45	F	5	Temporal	Throbbing	7	Visual				MWWOA	amt + prop	DIF	MWOA – Migraine without Aura
193	26	M	15	Temporal	Throbbing	24					CM	amt + prop	DIF	MWWOA - Migraine with and without Aura
194	14	F	18	Temporal	Pricking	3					MWOA	prop	DFI-6M	CM- Complications of Migraine
195	18	F	2	Occipital	Aching	4					MWOA	prop	DFI-6M	PM- Probable Migraine
196	52	F	18	Temporal	Throbbing	12	Visual				MWWOA	amt + prop	DFI-6M+	TTH-Tension type Head ache
197	19	M	20	Temporal	Throbbing	12					CM	amt + prop	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
198	27	F	3	Occipital	Diffuse	16	Visual				MWWOA	amt + prop	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
199	35	F	18	Temporal	Throbbing	12					MWOA	Amit	DFI-12M	PTTH- Probable Tension type Head ache
200	42	M	18	Frontal	Throbbing	12					MWOA	prop	DFI-6M+	CTTH-Chronic Tension type head ache
201	14	F	21	Temporal	Throbbing	24					CM	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
202	27	M	5MIN	Frontotemp	Aching	60					EPHC	Amitryptiline	DFI-6M	CCH – Chronic Cluster Headache
203	18	F	3	Temporal	Throbbing	8	Visual				MWWOA	Sod Valp	DFI-6M+	ECH –Episodic Cluster Headache
204	20	F	18	Temporal	Pricking	12		Yes			MWOA	amt + prop	DFI-12M	EPHC – Eposodic Paroxysmal Hemicrania
205	38	M	5	Temporal	Throbbing	12		Yes			MWOA	amt + prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
206	25	M	18	Parietal	Throbbing	24		Yes			MWOA	Amit	DIF	DFI-12m- Disease free interval for 12 months
207	19	F	11	Temporal	Pricking	8		Yes			MWOA	amt + prop	DFI-6M+	DFI-6m+ - Disease free interval 6months -12 months
208	36	F	18	Occipital	Throbbing	5	Sensory & Visual	Yes			MWA	prop	DFI-6M+	DFI-6m - Disease free interval less than 6 months
209	42	M	4	Temporal	Throbbing	12					MWOA	amt + prop	DFI-12M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
210	18	F	18	Frontal	Pricking	24		Yes			CM	amt + prop	DFI-6M+	MWA – Migraine with Aura
211	12	M	18	Temporal	Throbbing	14	Visual	Yes			MWWOA	amt + prop	DIF	MWOA – Migraine without Aura
212	31	F	24	Frontal	Throbbing	24		Yes			CM	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
213	24	F	3	Temporal	Throbbing	12	Visual	Yes			MWWOA	amt + prop	DFI-6M+	CM- Complications of Migraine
214	26	F	18	Frontal	Throbbing	12					MWOA	prop	DFI-6M	PM- Probable Migraine
215	30	F	18	Temporal	Pricking	24		Yes			MWOA	Amit	DFI-6M+	TTH-Tension type Head ache
216	34	M	12	Frontal	Throbbing	7	Visual				MWWOA	Amit	DFI-6M+	FETTH-Frequent Episodic Tension type Head ache
217	34	F	3	Frontal	Throbbing	12		Yes			MWOA	amt + prop	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
218	22	F	18	Frontal	Throbbing	12	Visual	Yes			MWA	prop	DFI-12M	PTTH- Probable Tension type Head ache
219	45	M	3	Frontotemp	Aching	30					SUNCT	Sod Valp	DIF	CTTH-Chronic Tension type head ache
220	18	M	18	Temporal	Throbbing	6		Yes			MWOA	amt + prop	DIF	TAC- Trigeminal Autonomic Cephalagias
221	22	F	14	Frontal	Throbbing	2	Visual				MWWOA	amt + prop	DFI-12M	CCH – Chronic Cluster Headache
222	26	M	15	Temporal	Throbbing	24		Yes			CM	amt + prop	DIF	ECH –Episodic Cluster Headache
223	15	F	18	Occipital	Pricking	4		Yes			MWOA	prop	DFI-12M	EPHC – Eposodic Paroxysmal Hemicrania
224	18	F	2	Temporal	Throbbing	14	Visual				MWWOA	amt + prop	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
225	38	M	12	Frontotemp	Aching	30					CPHC	Amitryptiline	DIF	DFI-12m- Disease free interval for 12 months
226	36	M	8	Temporal	Pricking	24		Yes			MWOA	Sod Valp	DFI-6M+	DFI-6m+ - Disease free interval 6months -12 months
227	29	M	18	Frontal	Throbbing	12			Basal Gang Calc		MWOA	prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
228	14	F	18	Temporal	Burning	24	Visual	Yes			MWA	prop	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
229	18	M	18	Occipital	Diffuse	3		Yes			MWOA	amt + prop	DFI-6M+	MWA – Migraine with Aura
230	53	F	24	Temporal	Diffuse	18		Yes			CM	amt + prop	DFI-6M+	MWOA – Migraine without Aura
231	22	F	14	Temporal	Throbbing	3	Visual	Yes	Calcif Granuloma		MWA	amt + prop	DIF	MWWOA - Migraine with and without Aura
232	34	F	12	Frontal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+	CM- Complications of Migraine
233	43	F	24	Temporal	Aching	8	visual	Yes			MWWOA	amt + prop	DFI-6M+	PM- Probable Migraine
234	39	M	3	Frontotemp	Aching	15					CPHC	Amitryptiline	DIF	TTH-Tension type Head ache
235	19	M	24	Temporal	Pricking	12					CM	prop	DFI-12M	FETTH-Frequent Episodic Tension type Head ache
236	52	F	18	Occipital	Throbbing	7		Yes			MWOA	Amit	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
237	38	F	18	Temporal	Throbbing	4					MWOA	amt + prop	DFI-12M	PTTH- Probable Tension type Head ache
238	44	F	24	FRONTAL	BAND	5					PTTH	Amitryptiline	DIF	CTTH-Chronic Tension type head ache
239	24	M	8	Frontal	Throbbing	8		Yes	Calcif Granuloma		CM	amt + prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
240	22	F	14	Temporal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-6M+	CCH – Chronic Cluster Headache
241	19	F	18	Frontal	Aching	3					MWOA	Amit	DFI-12M	ECH –Episodic Cluster Headache
242	17	F	24	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
243	29	F	12	Parietal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	CPHC – Chronic Paroxysmal Hemicrania
244	51	M	18	Temporal	Throbbing	2	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	DFI-12m- Disease free interval for 12 months
245	19	M	12	Parietal	Throbbing	20		Yes			CM	amt + prop	DFI-12M	DFI-6m+ - Disease free interval 6months -12 months
246	32	F	8	Temporal	Diffuse	5		Yes			MWOA	prop	DFI-6M+	DFI-6m - Disease free interval less than 6 months
247	26	M	24	Frontal	Throbbing	4	Visual				MWWOA	amt + prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
248	39	M	11	OCCIPITAL	ACHE	21					CTTH	Amitryptiline	NR	MWA – Migraine with Aura
249	50	F	24	Frontal	Throbbing	16					CM	amt + prop	DFI-6M+	MWOA – Migraine without Aura
250	22	F	5	Occipital	Throbbing	8		Yes			MWOA	Amit	DIF	MWWOA - Migraine with and without Aura
251	21	F	12	Temporal	Throbbing	5					MWOA	prop	DFI-6M	CM- Complications of Migraine
252	19	M	7	Temporal	Aching	3		Yes			MWOA	amt + prop	DIF	PM- Probable Migraine
253	36	M	20MIN	Frontal	Aching	6					PHSA	Amitryptiline	DIF	TTH-Tension type Head ache
254	24	F	12	Frontal	Throbbing	12			Calcif Granuloma		MWOA	amt + prop	DIF	FETTH-Frequent Episodic Tension type Head ache
255	35	F	9	PARIETAL	ACHE	17					CTTH	Amitryptiline	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
256	35	M	24	Temporal	Pricking	7	Visual	Yes			MWWOA	amt + prop	DFI-6M+	PTTH- Probable Tension type Head ache
257	16	F	24	Temporal	Throbbing	14			Calcif Granuloma		MWOA	prop	DFI-6M+	CTTH-Chronic Tension type head ache
258	17	F	18	Occipital	Pricking	1	Visual	Yes			MWWOA	prop	DFI-12M	TAC- Trigeminal Autonomic Cephalagias
259	15	F	5	Temporal	Throbbing	5		Yes			CM	prop	DFI-6M	CCH – Chronic Cluster Headache
260	37	F	12	Temporal	Throbbing	3					MWOA	amt + prop	DFI-6M+	ECH –Episodic Cluster Headache
261	53	F	5	OCCIPITAL	ACHE	15		YES			CTTH	Amitryptiline	DIF	EPHC – Eposodic Paroxysmal Hemicrania
262	50	M	12	Temporal	Throbbing	6		Yes			CM	amt + prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
263	13	M	8	Frontal	Throbbing	2					CM	amt + prop	DIF	DFI-12m- Disease free interval for 12 months
264	14	F	24	Frontal	Throbbing	3	Sensory				MWA	prop	DFI-6M+	DFI-6m+ - Disease free interval 6months -12 months
265	45	M	24	PARIETAL	PRICKING	2					IETTH	Amitryptiline	DFI-6M	DFI-6m - Disease free interval less than 6 months
266	15	F	18	Temporal	Throbbing	5		Yes			CM	prop	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
267	26	F	14	Temporal	Throbbing	22		Yes			MWOA	Amit	DFI-12M	MWA – Migraine with Aura
268	51	F	8	FRONTAL	ACHE	16					CTTH	Amitryptiline	DFI-6M+	MWOA – Migraine without Aura
269	59	M	30MIN	Frontotemp	Aching	4					HH	Amitryptiline	DIF	MWWOA - Migraine with and without Aura
270	41	F	7	Temporal	Throbbing	20		Yes	gliosis	Stroke	CM	amt + prop	DFI-6M	CM- Complications of Migraine
271	41	F	9	FRONTAL	ACHE	17					CTTH	Amitryptiline	DFI-6M	PM- Probable Migraine
272	16	F	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	TTH-Tension type Head ache
273	32	F	24	Occipital	Burning	2	Sensory	Yes			MWWOA	prop	DIF	FETTH-Frequent Episodic Tension type Head ache
274	28	M	24	Frontal	Throbbing	8		Yes			MWOA	amt + prop	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
275	39	M	11	OCCIPITAL	ACHE	21					CTTH	Amitryptiline	NR	PTTH- Probable Tension type Head ache
276	15	M	8	Temporal	Throbbing	2					MWOA	amt + prop	DFI-6M+	CTTH-Chronic Tension type head ache
277	32	F	9	Parietal	Throbbing	4	Visual	Yes			MWA	prop	DFI-6M	TAC- Trigeminal Autonomic Cephalagias
278	21	M	12	Temporal	Diffuse	4	Visual	Yes			MWWOA	amt + prop	DFI-6M+	CCH – Chronic Cluster Headache
279	17	F	12	Frontal	Throbbing	8		Yes	Calcif Granuloma		MWOA	prop	DFI-6M	ECH –Episodic Cluster Headache
280	43	M	10	TEMPORAL	ACHE	17		YES			CTTH	Amitryptiline	DIF	EPHC – Eposodic Paroxysmal Hemicrania
281	8	F	24	Temporal	Aching	5	Visual		Calcif Granuloma		MWA	prop	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
282	37	M	120	Frontotemp	Aching	1					NDPH	Amitryptiline	DIF	DFI-12m- Disease free interval for 12 months
283	36	F	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	DFI-6m+ - Disease free interval 6months -12 months
284	13	F	6	Occipital	Throbbing	3		Yes			MWOA	prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
285	15	F	18	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
286	44	F	7	TEMPORAL	ACHE	18					CTTH	Amitryptiline	DIF	MWA – Migraine with Aura
287	29	F	24	Temporal	Throbbing	2		Yes			MWOA	prop	DIF	MWOA – Migraine without Aura
288	31	F	24	Frontal	Throbbing	24		Yes			CM	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
289	29	M	12	Frontal	Throbbing	3	Visual	Yes			MWWOA	amt + prop	DFI-12M	CM- Complications of Migraine
290	42	F	13	FRONTAL	BAND	24					CTTH	Amitryptiline	DFI-6M+	PM- Probable Migraine
291	15	F	24	Temporal	Pricking	2			Calcif Granuloma		MWOA	amt + prop	DFI-6M+	TTH-Tension type Head ache
292	18	M	6	Temporal	Burning	4		Yes			MWOA	amt + prop	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
293	44	F	18	Parietal	Throbbing	2					MWOA	prop	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
294	20	F	6	Parietal	Throbbing	5		Yes			MWOA	amt + prop	DFI-6M	PTTH- Probable Tension type Head ache
295	24	M	24	Temporal	Throbbing	15		Yes			CM	amt + prop	DFI-6M	CTTH-Chronic Tension type head ache
296	15	F	12	Occipital	Pricking	4					MWOA	Amit	NR	TAC- Trigeminal Autonomic Cephalagias
297	26	F	12	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	CCH – Chronic Cluster Headache
298	45	F	36	TEMPORAL	BAND	9					IETTH	Amitryptiline	NR	ECH –Episodic Cluster Headache
299	26	M	15	Temporal	Throbbing	24		Yes			CM	amt + prop	DIF	EPHC – Eposodic Paroxysmal Hemicrania
300	42	M	96	Frontotemp	Aching	3					NDPH	Amitryptiline	DIF	CPHC – Chronic Paroxysmal Hemicrania
301	25	F	24	Occipital	Throbbing	3		Yes			MWOA	prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
302	30	F	12	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
303	32	F	24	Frontal	Throbbing	18		Yes			CM	amt + prop	DFI-12M	DFI-6m - Disease free interval less than 6 months
304	18	F	12	Temporal	Pricking	8					MWOA	amt + prop	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
305	47	M	12	Temporal	Throbbing	6		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	MWA – Migraine with Aura
306	22	F	14	Frontal	Throbbing	2	Visual				MWWOA	amt + prop	DFI-12M	MWOA – Migraine without Aura
307	15	F	24	Temporal	Aching	15		Yes			CM	amt + prop	DFI-6M	MWWOA - Migraine with and without Aura
308	12	F	18	Temporal	Throbbing	8		Yes			MWOA	Amit	DFI-6M	CM- Complications of Migraine
309	21	F	12	Frontal	Throbbing	6		Yes			MWOA	amt + prop	DFI-12M	PM- Probable Migraine
310	32	F	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	TTH-Tension type Head ache
311	25	F	6	Frontal	Throbbing	8	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
312	29	F	18	Occipital	Aching	6		Yes			MWOA	prop	DFI-12M	IETTH-Infrequent Episodic Tension type head ache
313	19	F	12	Frontal	Throbbing	4					MWOA	amt + prop	DFI-6M	PTTH- Probable Tension type Head ache
314	38	F	24	Temporal	Throbbing	3		Yes			MWOA	Amit	DFI-6M+	CTTH-Chronic Tension type head ache
315	57	F	13	FRONTAL	BAND	24					CTTH	Amitryptiline	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
316	22	M	6	Temporal	Throbbing	2	Sensory				MWWOA	prop	DFI-6M	CCH – Chronic Cluster Headache
317	18	M	18	Temporal	Pricking	4		Yes			MWOA	prop	DFI-6M+	ECH –Episodic Cluster Headache
318	35	F	12	Parietal	Throbbing	2		Yes			MWOA	Amit	NR	EPHC – Eposodic Paroxysmal Hemicrania
319	35	M	1MIN	FRONTAL	Stabbing	4					PSH	Amitryptiline	DIF	CPHC – Chronic Paroxysmal Hemicrania
320	44	F	24	FRONTAL	BAND	19					CTTH	Amitryptiline	DFI-6M	DFI-12m- Disease free interval for 12 months
321	34	F	24	Temporal	Diffuse	2		Yes			MWOA	prop	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
322	18	F	24	FRONTAL	BAND	7					FETTH	Amitryptiline	DIF	DFI-6m - Disease free interval less than 6 months
323	24	F	12	Temporal	Pricking	5	Visual				MWA	amt + prop	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
324	15	F	24	Frontal	Throbbing	20		Yes			CM	amt + prop	DFI-12M	MWA – Migraine with Aura
325	9	F	12	Frontal	Throbbing	6		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	MWOA – Migraine without Aura
326	39	M	11	OCCIPITAL	BAND	21					CTTH	Amitryptiline	NR	MWWOA - Migraine with and without Aura
327	20	F	6	Frontal	Throbbing	6		Yes			MWOA	Sod Valp	DIF	CM- Complications of Migraine
328	25	M	18	Temporal	Aching	4	Visual	Yes			MWWOA	Amit	DFI-12M	PM- Probable Migraine
329	17	M	24	Temporal	Throbbing	3		Yes			MWOA	amt + prop	DFI-6M	TTH-Tension type Head ache
330	39	M	11	OCCIPITAL	ACHE	21					CTTH	Amitryptiline	NR	FETTH-Frequent Episodic Tension type Head ache
331	15	M	6	Parietal	Throbbing	15			Calcif Granuloma		CM	amt + prop	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
332	37	F	18	Temporal	Throbbing	2	Sensory	Yes	Calcif Granuloma		MWWOA	prop	DIF	PTTH- Probable Tension type Head ache
333	19	F	6	Temporal	Throbbing	6					MWOA	prop	DIF	CTTH-Chronic Tension type head ache
334	25	F	24	Occipital	Aching	6		Yes			MWOA	amt + prop	DFI-12M	TAC- Trigeminal Autonomic Cephalagias
335	39	F	12	Frontal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	CCH – Chronic Cluster Headache
336	29	F	12	Temporal	Throbbing	6	Visual				MWWOA	prop	DFI-12M	ECH –Episodic Cluster Headache
337	12	F	24	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	EPHC – Eposodic Paroxysmal Hemicrania
338	44	F	126	Frontal	Throbbing	18		Yes	Calcif Granuloma		CM	prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
339	17	M	24	Temporal	Burning	2	Sensory & Visual	Yes			MWWOA	prop	DFI-6M+	DFI-12m- Disease free interval for 12 months
340	29	M	1MIN	FRONTAL	Stabbing	6					PSH	Amitryptiline	DIF	DFI-6m+ - Disease free interval 6months -12 months
341	22	M	18	Occipital	Throbbing	3		Yes			MWOA	amt + prop	DFI-12M	DFI-6m - Disease free interval less than 6 months
342	23	M	12	Frontal	Aching	16		Yes			CM	amt + prop	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
343	42	F	13	FRONTAL	ACHE	24					CTTH	Amitryptiline	DFI-6M+	MWA – Migraine with Aura
344	28	F	6	Frontal	Throbbing	15		Yes			MWOA	amt + prop	DFI-6M	MWOA – Migraine without Aura
345	36	F	12	Temporal	Pricking	5	Visual				MWWOA	Amit	NR	MWWOA - Migraine with and without Aura
346	19	F	24	Occipital	Throbbing	3		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-12M	CM- Complications of Migraine
347	49	F	9	FRONTAL	ACHE	17			calcific granuloma		CTTH	Amitryptiline	DFI-6M	PM- Probable Migraine
348	24	F	18	Temporal	Throbbing	2					MWOA	amt + prop	DFI-6M	TTH-Tension type Head ache
349	20	M	12	Frontal	Throbbing	5		Yes			MWOA	prop	DIF	FETTH-Frequent Episodic Tension type Head ache
350	28	F	12	Temporal	Aching	5		Yes			CM	amt + prop	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
351	22	F	24	Temporal	Pricking	3		Yes			MWOA	amt + prop	DFI-12M	PTTH- Probable Tension type Head ache
352	38	M	12	Occipital	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	CTTH-Chronic Tension type head ache
353	41	M	15	FRONTAL	ACHE	12		YES	calcific granuloma		IETTH	Amitryptiline	DIF	TAC- Trigeminal Autonomic Cephalagias
354	52	M	16	FRONTAL	ACHE	10		YES			FETTH	Amitryptiline	DFI-6M	CCH – Chronic Cluster Headache
355	18	F	24	Temporal	Throbbing	20	Visual	Yes			MWWOA	prop	DIF	ECH –Episodic Cluster Headache
356	32	M	96	Frontotemp	Aching	2					NDPH	Amitryptiline	DIF	EPHC – Eposodic Paroxysmal Hemicrania
357	21	F	24	Frontal	Throbbing	3		Yes			MWOA	amt + prop	DFI-12M	CPHC – Chronic Paroxysmal Hemicrania
358	17	M	12	Parietal	Throbbing	5	Visual		Calcif Granuloma		MWA	prop	DIF	DFI-12m- Disease free interval for 12 months
359	48	M	16	FRONTAL	ACHE	10		YES	calcific granuloma		FETTH	Amitryptiline	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
360	32	F	12	Temporal	Pricking	3		Yes			MWOA	Amit	DFI-6M+	DFI-6m - Disease free interval less than 6 months
361	18	F	6	Occipital	Throbbing	2	Visual	Yes			MWWOA	prop	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
362	34	F	6	Temporal	Pricking	2					MWOA	Amit	DFI-6M	MWA – Migraine with Aura
363	7	F	12	Temporal	Pricking	6	Visual				MWA	prop	DIF	MWOA – Migraine without Aura
364	19	M	20	Temporal	Throbbing	12					CM	amt + prop	DFI-6M+	MWWOA - Migraine with and without Aura
365	52	F	2	Temporal	Throbbing	12	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M+	CM- Complications of Migraine
366	18	F	12	Frontal	Throbbing	15		Yes	Calcif Granuloma		CM	amt + prop	DFI-12M	PM- Probable Migraine
367	24	F	24	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DFI-6M	TTH-Tension type Head ache
368	36	F	12	Frontal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
369	18	F	24	Temporal	Throbbing	20	Visual	Yes			MWWOA	prop	DIF	IETTH-Infrequent Episodic Tension type head ache
370	29	F	24	Frontal	Throbbing	18		Yes			CM	amt + prop	DFI-6M	PTTH- Probable Tension type Head ache
371	27	M	6	Occipital	Diffuse	6	Visual	Yes			MWWOA	prop	DIF	CTTH-Chronic Tension type head ache
372	22	M	6	Occipital	Throbbing	4	Sensory				MWWOA	amt + prop	DFI-12M	TAC- Trigeminal Autonomic Cephalagias
373	24	F	12	Temporal	Pricking	6		Yes			MWOA	amt + prop	DFI-6M	CCH – Chronic Cluster Headache
374	36	F	24	Temporal	Throbbing	2	Visual	Yes			MWWOA	Amit	NR	ECH –Episodic Cluster Headache
375	36	F	15	Frontal	Throbbing	22					CM	amt + prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
376	32	F	12	Temporal	Throbbing	11	Sensory	Yes	Calcif Granuloma		MWWOA	amt + prop	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
377	28	F	24	Frontal	Aching	8		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-6M	DFI-12m- Disease free interval for 12 months
378	18	M	12	Temporal	Throbbing	2		Yes			MWOA	amt + prop	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
379	15	F	12	Temporal	Pricking	18		Yes			CM	amt + prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
380	28	M	6	Temporal	Throbbing	1	Visual	Yes			MWWOA	amt + prop	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
381	28	F	24	FRONTAL	BAND	5			calcific granuloma		PTTH	Amitryptiline	DIF	MWA – Migraine with Aura
382	19	F	6	Temporal	Burning	4		Yes			MWOA	prop	DFI-12M	MWOA – Migraine without Aura
383	19	M	24	Frontal	Pricking	4		Yes			MWOA	Amit	DFI-6M+	MWWOA - Migraine with and without Aura
384	48	M	16	FRONTAL	BAND	10		YES			FETTH	Amitryptiline	DFI-6M	CM- Complications of Migraine
385	33	F	24	Frontal	Throbbing	3		Yes	Calcif Granuloma		MWOA	Amit	DFI-6M	PM- Probable Migraine
386	53	F	12	Occipital	Throbbing	6	Visual				MWWOA	Amit	DFI-6M	TTH-Tension type Head ache
387	36	F	6	FRONTAL	ACHE	4		YES			PTTH	Amitryptiline	DIF	FETTH-Frequent Episodic Tension type Head ache
388	27	M	24	Temporal	Throbbing	3		Yes			MWOA	prop	DIF	IETTH-Infrequent Episodic Tension type head ache
389	15	F	24	Frontal	Throbbing	4					MWOA	amt + prop	DFI-6M	PTTH- Probable Tension type Head ache
390	33	F	24	Temporal	Throbbing	22		Yes			CM	amt + prop	DFI-12M	CTTH-Chronic Tension type head ache
391	16	M	12	Temporal	Throbbing	6		Yes			MWOA	Amit	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
392	47	F	24	FRONTAL	ACHE	10					FETTH	Amitryptiline	DFI-6M	CCH – Chronic Cluster Headache
393														ECH –Episodic Cluster Headache
394	26	F	24	Parietal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DFI-12M	EPHC – Eposodic Paroxysmal Hemicrania
395	18	F	6	Temporal	Pricking	2		Yes			MWOA	amt + prop	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
396	32	M	24	Temporal	Aching	6	Visual	Yes	Calcif Granuloma		MWA	prop	DIF	DFI-12m- Disease free interval for 12 months
397	10	F	12	Temporal	Throbbing	4		Yes			MWOA	Amit	DFI-6M+	DFI-6m+ - Disease free interval 6months -12 months
398	42	F	13	FRONTAL	ACHE	24					CTTH	Amitryptiline	DFI-6M+	DFI-6m - Disease free interval less than 6 months
399	36	F	6	Temporal	Aching	3	Visual	Yes	Calcif Granuloma		MWA	prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
400	35	M	24	Frontal	Aching	15		Yes			CM	amt + prop	DFI-12M	MWA – Migraine with Aura
401	45	M	24	PARIETAL	ACHE	2					IETTH	Amitryptiline	DFI-6M	MWOA – Migraine without Aura
402	18	F	6	Occipital	Throbbing	2	Visual	Yes			MWWOA	prop	DFI-6M+	MWWOA - Migraine with and without Aura
403	42	F	24	Frontal	Throbbing	5					MWOA	amt + prop	DIF	CM- Complications of Migraine
404	16	F	12	Temporal	Pricking	3		Yes	Calcif Granuloma		MWOA	Amit	DFI-12M	PM- Probable Migraine
405	30	M	6	Temporal	Throbbing	2	Sensory & Visual		Calcif Granuloma		MWA	amt + prop	DFI-6M	TTH-Tension type Head ache
406	20	M	24	Frontal	Aching	8		Yes			MWOA	amt + prop	DFI-12M	FETTH-Frequent Episodic Tension type Head ache
407	15	F	12	Temporal	Pricking	18		Yes			CM	amt + prop	DFI-6M	IETTH-Infrequent Episodic Tension type head ache
408	52	F	6	Temporal	Throbbing	6	Sensory	Yes			MWWOA	amt + prop	DFI-6M	PTTH- Probable Tension type Head ache
409	28	F	24	FRONTAL	BAND	5			calcific granuloma		PTTH	Amitryptiline	DIF	CTTH-Chronic Tension type head ache
410	18	F	12	Parietal	Throbbing	4		Yes			MWOA	prop	DIF	TAC- Trigeminal Autonomic Cephalagias
411	16	F	24	Temporal	Pricking	8	Visual				MWWOA	Amit	DFI-6M	CCH – Chronic Cluster Headache
412	41	M	20	FRONTAL	ACHE	20					CTTH	Amitryptiline	DFI-6M	ECH –Episodic Cluster Headache
413	24	M	24	Frontal	Throbbing	18		Yes	Calcif Granuloma		CM	amt + prop	DFI-12M	EPHC – Eposodic Paroxysmal Hemicrania
414	40	F	6	Parietal	Throbbing	10		Yes			MWOA	amt + prop	DFI-6M	CPHC – Chronic Paroxysmal Hemicrania
415	25	M	6	Temporal	Throbbing	4	Visual	Yes			MWWOA	amt + prop	DFI-6M	DFI-12m- Disease free interval for 12 months
416	30	F	12	Temporal	Aching	2	Visual				MWA	prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
417	26	M	12	Frontal	Throbbing	4		Yes	Calcif Granuloma		MWOA	amt + prop	DFI-12M	DFI-6m - Disease free interval less than 6 months
418	56	F	4	FRONTAL	ACHE	8		YES			FETTH	Amitryptiline	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
419	18	F	5	Frontal	Throbbing	20		Yes			CM	amt + prop	DFI-6M	MWA – Migraine with Aura
420	20	F	12	Temporal	Throbbing	2		Yes			MWOA	prop	DIF	MWOA – Migraine without Aura
421	51	F	24	Occipital	Diffuse	2		Yes			MWOA	Amit	DFI-6M+	MWWOA - Migraine with and without Aura
422	44	F	24	PARIETAL	ACHE	16		YES			CTTH	Amitryptiline	DFI-12M	CM- Complications of Migraine
423	12	M	6	Temporal	Throbbing	1		Yes			MWOA	prop	DFI-12M	PM- Probable Migraine
424	19	M	12	Temporal	Throbbing	4					MWOA	amt + prop	DFI-6M	TTH-Tension type Head ache
425	26	F	7	TEMPORAL	ACHE	18					CTTH	Amitryptiline	DIF	FETTH-Frequent Episodic Tension type Head ache
426	26	F	12	Temporal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DIF	IETTH-Infrequent Episodic Tension type head ache
427														PTTH- Probable Tension type Head ache
428	42	F	22	FRONTAL	BAND	15					CTTH	Amitryptiline	DIF	CTTH-Chronic Tension type head ache
429	15	F	24	Frontal	Throbbing	15					CM	amt + prop	DIF	TAC- Trigeminal Autonomic Cephalagias
430	33	F	6	Occipital	Pricking	2		Yes			MWOA	prop	DFI-6M+	CCH – Chronic Cluster Headache
431	34	M	4	TEMPORAL	ACHE	16		YES			CTTH	Amitryptiline	DFI-12M	ECH –Episodic Cluster Headache
432	27	M	6	Occipital	Throbbing	3	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	EPHC – Eposodic Paroxysmal Hemicrania
433	35	F	12	Temporal	Burning	4	Sensory				MWWOA	amt + prop	DIF	CPHC – Chronic Paroxysmal Hemicrania
434	40	M	24	Occipital	Throbbing	4		Yes			MWOA	amt + prop	DFI-6M	DFI-12m- Disease free interval for 12 months
435	45	M	24	FRONTAL	ACHE	2					IETTH	Amitryptiline	DFI-6M	DFI-6m+ - Disease free interval 6months -12 months
436	18	F	24	Temporal	Throbbing	6		Yes			MWOA	prop	DIF	DFI-6m - Disease free interval less than 6 months
437	38	M	9	FRONTAL	BAND	2		YES	calcific granuloma		PTTH	Amitryptiline	DFI-6M+	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
438	30	F	6	Occipital	Throbbing	7	Sensory		Calcif Granuloma		MWWOA	prop	DFI-6M+	MWA – Migraine with Aura
439	25	F	12	Temporal	Pricking	8		Yes			MWOA	Amit	DFI-6M+	MWOA – Migraine without Aura
440	53	M	20	FRONTAL	ACHE	20					CTTH	Amitryptiline	DFI-6M	MWWOA - Migraine with and without Aura
441	23	M	12	Frontal	Aching	16		Yes			CM	amt + prop	DFI-6M	CM- Complications of Migraine
442	35	F	24	Temporal	Throbbing	8		Yes			MWOA	amt + prop	DFI-12M	PM- Probable Migraine
443	33	M	24	FRONTAL	ACHE	15					CTTH	Amitryptiline	DFI-6M+	TTH-Tension type Head ache
444	26	F	12	Frontal	Throbbing	8		Yes			MWOA	Amit	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
445	56	F	4	FRONTAL	ACHE	8		YES			FETTH	Amitryptiline	DFI-6M+	IETTH-Infrequent Episodic Tension type head ache
446	22	F	6	Frontal	Aching	4	visual	Yes	Calcif Granuloma		MWA	prop	DIF	PTTH- Probable Tension type Head ache
447	25	F	24	Parietal	Throbbing	3	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	CTTH-Chronic Tension type head ache
448	17	F	6	Temporal	Pricking	2		Yes	Calcif Granuloma		MWOA	amt + prop	DIF	TAC- Trigeminal Autonomic Cephalagias
449	29	M	3	FRONTAL	BAND	20					CTTH	Amitryptiline	DFI-6M	CCH – Chronic Cluster Headache
450	28	F	24	Frontal	Throbbing	5	Visual	Yes			MWA	prop	DFI-12M	ECH –Episodic Cluster Headache
451	14	M	6	Parietal	Throbbing	3		Yes			MWOA	prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
452	20	F	12	Temporal	Pricking	8		Yes			MWOA	amt + prop	DFI-12M	CPHC – Chronic Paroxysmal Hemicrania
453	26	M	24	Frontal	Throbbing	6		Yes			MWOA	Amit	DFI-6M+	DFI-12m- Disease free interval for 12 months
454	15	F	6	Occipital	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	DFI-6m+ - Disease free interval 6months -12 months
455	26	F	24	Temporal	Pricking	2	Visual	Yes			MWWOA	amt + prop	DIF	DFI-6m - Disease free interval less than 6 months
456	19	F	6	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DIF	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
457	30	F	24	Temporal	Aching	11					MWOA	amt + prop	DFI-12M	MWA – Migraine with Aura
458	25	M	12	Frontal	Throbbing	2	Visual	Yes			MWWOA	amt + prop	DIF	MWOA – Migraine without Aura
459	38	M	24	Temporal	Diffuse	20		Yes			CM	amt + prop	DIF	MWWOA - Migraine with and without Aura
460	15	F	6	Temporal	Throbbing	1	Visual				MWWOA	prop	DFI-12M	CM- Complications of Migraine
461	24	F	12	Frontal	Throbbing	4		Yes			MWOA	amt + prop	DIF	PM- Probable Migraine
462	18	F	9	FRONTAL	ACHE	17			calcific granuloma		CTTH	Amitryptiline	DFI-6M	TTH-Tension type Head ache
463	28	F	24	Temporal	Aching	2	Visual	Yes			MWWOA	amt + prop	DFI-12M	FETTH-Frequent Episodic Tension type Head ache
464	26	F	24	Temporal	Throbbing	4		Yes			MWOA	amt + prop	DFI-12M	IETTH-Infrequent Episodic Tension type head ache
465	40	M	24	Temporal	Pricking	8		Yes	Calcif Granuloma		MWOA	Amit	DFI-12M	PTTH- Probable Tension type Head ache
466	34	M	4	TEMPORAL	ACHE	16		YES			CTTH	Amitryptiline	DFI-12M	CTTH-Chronic Tension type head ache
467	40	F	6	Parietal	Throbbing	7		Yes			PM	prop	DFI-6M+	TAC- Trigeminal Autonomic Cephalagias
468	18	F	6	Temporal	Throbbing	9	Visual	Yes			MWWOA	amt + prop	DIF	CCH – Chronic Cluster Headache
469	14	F	24	Temporal	Throbbing	4	Visual	Yes			MWWOA	amt + prop	DFI-12M	ECH –Episodic Cluster Headache
470	26	F	12	Frontal	Aching	4					MWOA	amt + prop	DIF	EPHC – Eposodic Paroxysmal Hemicrania
471	43	F	32	FRONTAL	ACHE	17					CTTH	Amitryptiline	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
472	19	M	12	Temporal	Throbbing	8		Yes			MWOA	amt + prop	DFI-12M	DFI-12m- Disease free interval for 12 months
473	22	F	6	Temporal	Burning	7					MWOA	amt + prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
474	48	M	16	FRONTAL	ACHE	10		YES	calcific granuloma		FETTH	Amitryptiline	DFI-6M	DFI-6m - Disease free interval less than 6 months
475	28	F	6	Occipital	Throbbing	4	Sensory	Yes			MWWOA	prop	DFI-12M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
476	13	M	24	Frontal	Throbbing	8		Yes			MWOA	amt + prop	DIF	MWA – Migraine with Aura
477	28	F	24	FRONTAL	BAND	5			calcific granuloma		PTTH	Amitryptiline	DIF	MWOA – Migraine without Aura
478	30	F	6	Occipital	Throbbing	7	Sensory		Calcif Granuloma		MWWOA	prop	DFI-6M+	MWWOA - Migraine with and without Aura
479	15	F	6	Temporal	Pricking	9		Yes			PM	amt + prop	DIF	CM- Complications of Migraine
480	34	M	4	TEMPORAL	ACHE	16		YES			CTTH	Amitryptiline	DFI-12M	PM- Probable Migraine
481	19	M	24	Frontal	Pricking	4		Yes			MWOA	Amit	DFI-6M+	TTH-Tension type Head ache
482	25	F	7	TEMPORAL	ACHE	27		YES			CTTH	Amitryptiline	DFI-12M	FETTH-Frequent Episodic Tension type Head ache
483	27	F	12	Frontal	Throbbing	2	Visual	Yes	Calcif Granuloma		MWWOA	amt + prop	DIF	IETTH-Infrequent Episodic Tension type head ache
484	22	F	12	Parietal	Throbbing	5	Visual	Yes			MWWOA	amt + prop	DIF	PTTH- Probable Tension type Head ache
485	38	M	24	Temporal	Diffuse	20		Yes			CM	amt + prop	DIF	CTTH-Chronic Tension type head ache
486	38	F	20	OCCIPITAL	ACHE	3			calcific granuloma		IETTH	Amitryptiline	DFI-6M	TAC- Trigeminal Autonomic Cephalagias
487	22	F	15	FRONTAL	ACHE	12		YES	calcific granuloma		IETTH	Amitryptiline	DIF	CCH – Chronic Cluster Headache
488	30	F	6	Frontal	Throbbing	14		Yes			MWOA	amt + prop	DFI-12M	ECH –Episodic Cluster Headache
489	31	F	24	Frontal	Throbbing	24		Yes			CM	amt + prop	DFI-6M+	EPHC – Eposodic Paroxysmal Hemicrania
490	40	F	6	Parietal	Throbbing	7		Yes			PM	prop	DFI-6M+	CPHC – Chronic Paroxysmal Hemicrania
491	24	F	9	FRONTAL	ACHE	17			calcific granuloma		CTTH	Amitryptiline	DFI-6M	DFI-12m- Disease free interval for 12 months
492	17	M	24	Temporal	Pricking	3	Visual	Yes	Calcif Granuloma		MWWOA	prop	DIF	DFI-6m+ - Disease free interval 6months -12 months
493	24	F	6	Temporal	Throbbing	9	Sensory & Visual	Yes			MWWOA	amt + prop	DFI-6M	DFI-6m - Disease free interval less than 6 months
494	47	F	24	FRONTAL	ACHE	10			calcific granuloma		FETTH	Amitryptiline	DFI-6M	NR – No response

NO	AGE	SEX	DURATION IN HRS	LOCATION	CHARACTER	FREQUENCY / MONTH	AURA	FAMILY HISTORY	CTSCAN - BRAIN	COMPLICATIONS	DIAGNOSIS	PROPHYLAX	RESPONSE	ABBREVIATIONS
495	36	F	24	Temporal	Aching	15		Yes			MWOA	amt + prop	DIF	MWA – Migraine with Aura
496	22	F	12	Frontal	Throbbing	12					PM	amt + prop	DFI-6M	MWOA – Migraine without Aura
497	56	F	13	FRONTAL	ACHE	24					CTTH	Amitryptiline	DFI-6M+	MWWOA - Migraine with and without Aura
498	24	F	24	FRONTAL	BAND	7			calcific granuloma		FETTH	Amitryptiline	DIF	CM- Complications of Migraine
499	26	F	7	TEMPORAL	ACHE	18					CTTH	Amitryptiline	DIF	PM- Probable Migraine
500	14	M	24	Temporal	Throbbing	3					MWOA	prop	DFI-6M	TTH-Tension type Head ache
501	29	M	3	FRONTAL	BAND	20					CTTH	Amitryptiline	DFI-6M	FETTH-Frequent Episodic Tension type Head ache
502	18	M	6	Occipital	Throbbing	4		Yes			MWOA	prop	DIF	IETTH-Infrequent Episodic Tension type head ache

International Headache Society Classification of Migraine (2004)

- 1.1 Migraine without aura
- 1.2 Migraine with aura
  - 1.2.1 Typical aura with migraine headache
  - 1.2.2 Typical aura with non-migraine headache
  - 1.2.3 Typical aura without headache
  - 1.2.4 Familial hemiplegic migraine (FHM)
  - 1.2.5 Sporadic hemiplegic migraine
  - 1.2.6 Basilar-type migraine
- 1.3 Childhood periodic syndromes that are commonly precursors of migraine
  - 1.3.1 Cyclical vomiting
  - 1.3.2 Abdominal migraine
  - 1.3.3 Benign paroxysmal vertigo of childhood
- 1.4 Retinal migraine
- 1.5 Complications of migraine
  - 1.5.1 Chronic migraine
  - 1.5.2 Status migrainosus
  - 1.5.3 Persistent aura without infarction
  - 1.5.4 Migrainous infarction
  - 1.5.5 Migraine-triggered seizures
- 1.6 Probable migraine
  - 1.6.1 Probable migraine without aura
  - 1.6.2 Probable migraine with aura
  - 1.6.5 Probable chronic migraine

International Headache Society Diagnosis Criteria for Migraine

1.1 Migraine without aura: This is a clinical syndrome characterised by recurrent headache disorder manifesting in attacks lasting 4-72 hours, with typical headache of unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia. The diagnostic criteria include: (A) At least 5 attacks fulfilling criteria B-D; (B) Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated); (C) Headache has at least two of the following characteristics: 1. unilateral location, 2. pulsating quality, 3. moderate or severe pain intensity, 4. aggravation by or causing avoidance of routine physical activity (*eg*, walking or climbing stairs); (D) During headache at least one of the following: 1. nausea and/or vomiting, 2. photophobia and phonophobia; and (E) Not attributed to another disorder

1.2 Migraine with aura: This entity previously termed classical migraine is a recurrent disorder of reversible focal neurological symptoms that develops gradually over 5-20 minutes and last for less than 60 minutes,. Headache with features of migraine without aura usually follows the aura symptoms, and less commonly, headache lacks migrainous features or is completely absent. The aura is a complex of neurological symptoms that occurs just before or at the onset of migraine headache. Most patients with migraine have exclusively attacks without aura. Many patients who have frequent attacks with aura may also have attacks without aura. Diagnostic criteria include [A] At least 2 attacks fulfilling criterion B [B] Migraine aura fulfilling criteria B and C for one of the subforms 1.2.1-1.2.6 & [C] Not attributed to another disorder. Premonitory symptoms occur hours to a day or two before a migraine attack (with or without aura), that include various combinations of fatigue, difficulty in concentrating, neck stiffness,

sensitivity to light or sound, nausea, blurred vision, yawning and pallor. Auras with similar feature are also described in other well-defined headache types like cluster headache.

1.2.1 Migraine with typical aura: Typical aura consists of visual and/or sensory and/or speech symptoms. Gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is associated with a headache fulfilling criteria for Migraine without aura. Diagnostic criteria include (A) At least 2 attacks fulfilling criteria B–D; (B) Aura consisting of at least one of the following, but no motor weakness: 1. fully reversible visual symptoms including positive features (eg, flickering lights, spots or lines) and/or negative features (ie, loss of vision), 2. fully reversible sensory symptoms including positive features (ie, pins and needles) and/or negative features (ie, numbness), 3. fully reversible dysphasic speech disturbance; (C) At least two of the following: 1. homonymous visual symptoms and/or unilateral sensory symptoms 2. at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes, 3. each symptom lasts ≥5 and ≤60 minutes; (D) Headache fulfilling criteria B-D for 1.1 Migraine without aura begins during the aura or follows aura within 60 minutes; (E) Not attributed to another disorder. This is the most common migraine syndrome associated with aura, the diagnosis of which is evident with careful history alone, but rarely carotid dissection, AV malformation and seizure may mimic this condition. Aura include visual aura, the most common type that often presents as a fortification spectrum or scotoma without positive phenomena; sensory aura, that presents as pins and needles moving slowly from the point of origin or numbness and speech disturbances like dysphasia. Symptoms usually follow one another in succession beginning with visual, then sensory symptoms and dysphasia.

1.2.2 : Typical aura with non-migraine headache: Typical aura consisting of visual and/or sensory and/or speech symptoms with gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is associated with a headache that does not fulfil criteria for 1.1 Migraine without aura. Diagnostic criteria includes all patterns as for Migraine with typical aura from A – C and a Headache that does not fulfil the criteria B-D set for 1.1 *Migraine without aura,* that begins during the aura or follows aura within 60 minutes. In the absence of headache fulfilling the criteria for 1.1 *Migraine without aura,* precise diagnosis of aura and its distinction from more serious diseases like transient ischemic attack becomes more important.

1.2.3: Typical aura without headache: Typical aura consisting of visual and/or sensory symptoms with or without speech symptoms. Gradual development, duration no longer than one hour, a mix of positive and negative features and complete reversibility characterise the aura which is not associated with headache. A small number of patients have typical aura without headache are commonly patients with Typical aura with migraine who on becoming older have lost migraine characteristics, even though auras continue. Diagnostic criteria includes all patterns as for Migraine with typical aura from A – C without Headache during the aura or following aura within 60 minutes.

1.2.4: Familial hemiplegic migraine (FHM): Migraine with aura including motor weakness and at least one first- or second-degree relative has migraine aura including motor weakness. Specific genetic subtypes of FHM have been identified: (1) FHM1 - mutations in CACNA1A gene on chromosome 19 (2) FHM2 - mutations in the ATP1A2 gene on chromosome1, of these FHM1 very often has basilar-type symptoms in addition to the typical aura while headache is virtually always present.

1.2.5 Sporadic hemiplegic migraine: Migraine with aura including motor weakness without first or second-degree relative having an aura including motor weakness. The attacks have the same clinical characteristics as those in FHM.

1.2.6 Basilar-type migraine: Previously termed as Basilar artery migraine or basilar migraine, this entity is migraine with aura symptoms clearly originating from the brainstem and/or from both hemispheres simultaneously affected, but no motor weakness. Diagnostic criteria include: A. At least 2 attacks fulfilling criteria B-D; B. Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness: [1] dysarthria [2] vertigo [3] tinnitus [4] hypacusia [5] diplopia [6] visual symptoms simultaneously in both temporal and nasal fields of both eyes [7] ataxia [8] decreased level of consciousness [9] simultaneously bilateral paraesthesias; C. At least one of the following: [1] at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes [2] each aura symptom lasts ≥5 and ≤60 minutes; D.Headache fulfilling criteria B-D for 1.1 *Migraine without aura* begins during the aura or follows aura within 60 minutes; E. Not attributed to another disorder.

1.3 Childhood periodic syndromes that are commonly precursors of migraine.

1.3.1 Cyclical vomiting: Recurrent episodic attacks, usually stereotypical in the individual patient, of vomiting and intense nausea. Attacks are associated with pallor and lethargy. There is complete resolution of symptoms between attacks. Cyclical vomiting is a self-limiting episodic condition of childhood, with periods of complete normality between episodes

1.3.2 Abdominal migraine: An idiopathic recurrent disorder seen mainly in children and characterised by episodic midline abdominal pain manifesting in attacks lasting 1-72 hours with normality between episodes. The pain is of moderate to severe intensity and associated with vasomotor symptoms, nausea and vomiting.

1.3.3 Benign paroxysmal vertigo of childhood: This probably heterogeneous disorder is characterised by recurrent brief episodic attacks of vertigo occurring without warning and resolving spontaneously in otherwise healthy children.

1.4 Retinal migraine: Repeated attacks of monocular visual disturbance, including scintillations, scotomata or blindness, associated with migraine headache.

1.5 Complications of migraine

1.5.1 Chronic migraine: Migraine headache occurring on 15 or more days per month for more than 3 months in the absence of medication overuse. Diagnostic criteria: [A] Headache fulfilling criteria C and D for 1.1 Migraine without aura on ≥15 days/month for >3 months. [B] Not attributed to another disorder. Most cases of chronic migraine start as 1.1 Migraine without aura and therefore, may be regarded as a complication of episodic migraine. As chronicity develops, headache tends to lose its attack-wise (episodic) presentation although it has not been clearly demonstrated that this is always so. Therefore, the default rule is to code such patients according to the antecedent migraine subtype plus probable chronic migraine plus probable medication-overuse headache.

1.5.2 Status migrainosus: A debilitating migraine attack lasting for more than 72 hours. Diagnostic criteria: [A] The present attack in a patient with 1.1 Migraine without aura is

typical of previous attacks except for its duration [B] Headache has both of the following features: (1) unremitting for >72 hours (2) severe intensity [C] Not attributed to another disorder.

1.5.3 Persistent aura without infarction: Aura symptoms persist for more than 1 week without radiographic evidence of infarction. Diagnostic criteria: [A].The present attack in a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for >1 week [B]. Not attributed to another disorder. Persisting aura symptoms are rare but well documented. They are often bilateral and may last for months or years. Reliably effective treatment is not known though acetazolamide and valproic acid have helped in a few cases. It is important to exclude posterior leukoencephalopathy by diffusion MRI and Migrainous infarction.

1.5.4 Migrainous infarction: One or more migrainous aura symptoms associated with an ischaemic brain lesion in appropriate territory demonstrated by neuroimaging. Diagnostic criteria: [A] The present attack in a patient with 1.2 Migraine with aura is typical of previous attacks except that one or more aura symptoms persists for >60 minutes [B] Neuroimaging demonstrates ischaemic infarction in a relevant area. [C] Not attributed to another disorder. Ischaemic stroke in a migraine sufferer may be categorised as cerebral infarction of other cause coexisting with migraine, cerebral infarction of other cause presenting with symptoms resembling migraine with aura, or cerebral infarction occurring during the course of a typical migraine with aura attack. Only the last fulfils criteria for 1.5.4 Migrainous infarction. Increased risk for stroke in migraine patients has been demonstrated in women under age 45 in several studies. Evidence for an association between migraine and stroke in older women and in men is inconsistent.

1.5.5 Migraine-triggered seizure: A seizure triggered by a migraine aura. Diagnostic criteria: [A] Migraine fulfilling criteria for 1.2 Migraine with aura [B] A seizure fulfilling diagnostic criteria for one type of epileptic attack occurs during or within 1 hour after a migraine aura. Migraine and epilepsy are highly comorbid conditions probably sharing the same pathophysiology, but the nature of their association is unclear. Migralepsy is the term used when a seizure occurs during or within 1 hour of a typical migraine aura attack. Reversible brain MRI abnormalities have been reported in a patient with migraine-triggered seizure, possibly due to supratentorial focal cerebral edema. Electroencephalogram (EEG) findings are usually normal interictal, although various abnormalities, mainly diffuse slowing, have been reported in migraineurs. [23]

Symptom	**Migraine**	**Epilepsy**
Duration of aura	15–60 min	Brief, often <1 min
Automatisms	Unusual	Frequent for complex partial seizures
Gastrointestinal aura	Abdominal pain (rare) Nausea (common)	‘‘Butterflies’’—rising epigastric sensation
Visual disturbances	Positive/negative	Complex visual phenomenon
Paresthesias	Common (5–60 min)	Common (seconds to minutes)
Altered consciousness	Usually responsive	Often unresponsive
Olfactory	Very uncommon	More common
Aphasia	Uncommon	Common
D_ej_a vu	Rare	Common

1.6 Probable migraine: Previously termed as a migrainous disorder, attacks and/or headache missing one of the features needed to fulfil the criteria for the disorders coded above.

IHS CLASSIFICATION OF TENSION-TYPE HEADACHES –ICHD -2

2.1 Infrequent episodic tension-type headache

2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness

2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness

2.2 Frequent episodic tension-type headache

2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness

2.2.2 Frequent episodic tension-type headache not associated with pericranial tenderness

2.3 Chronic tension-type headache

2.3.1 Chronic tension-type headache associated with pericranial tenderness

2.3.2 Chronic tension-type headache not associated with pericranial tenderness

2.4 Probable tension-type headache

2.4.1 Probable infrequent episodic tension-type headache

2.4.2 Probable frequent episodic tension-type headache

2.4.3 Probable chronic tension-type headache

IHS DIAGNOSTIC CRITERIA FOR TENSION TYPE HEADACHES

2.1 Infrequent episodic tension-type headache:

Infrequent episodes of headache lasting minutes to days, the pain being typically bilateral, pressing or tightening in quality and of mild to moderate intensity, which does not worsen with routine physical activity, without nausea but photophobia or phonophobia may be present.

Diagnostic criteria include (A) At least 10 episodes occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B-D (B) Headache lasting from 30 minutes to 7 days (C) Headache has at least two of the following characteristics: (1) bilateral location (2) pressing/tightening (non-pulsating) quality (3) mild or moderate intensity (4) not aggravated by routine physical activity such as walking or climbing stairs (D) Both of the following: (1) no nausea or vomiting (anorexia may occur) (2) no more than one of photophobia or phonophobia (E) Not attributed to another disorder. [6]

2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness: Diagnostic criteria includes (A) Episodes fulfilling criteria A-E for 2.1 Infrequent episodic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness: Diagnostic criteria includes: (A) Episodes fulfilling criteria A-E for 2.1 Infrequent episodic tension-type headache (B) No increased pericranial tenderness. Increased pericranial tenderness recorded by manual palpation is the most significant abnormal finding in patients with tension-type headache. The tenderness increases with the intensity and frequency of headache and is further increased during actual headache. The diagnostic value of EMG and pressure algometry is limited Palpation is a useful guide for the treatment strategy. It also adds value and credibility to the explanations given to the patient.

2.2 Frequent episodic tension-type headache:

Frequent episodes of headache lasting minutes to days. The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, and it does not worsen with routine physical activity. There is no nausea but photophobia or phonophobia may be present. Diagnostic criteria include (A) At least 10 episodes occurring on ≥1 but <15 days per month for at least 3 months (≥12 and <180 days per year) and fulfilling criteria B-D (B) Headache lasting from 30 minutes to 7 days, (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity, 4. not aggravated by routine physical activity such as walking or climbing stairs, (D) Both of the following: 1. no nausea or vomiting (anorexia may occur), 2. no more than one of photophobia or phonophobia, (E) Not attributed to another disorder. Frequent tension-type headache often coexists with migraine without aura. Coexisting tension-type headache in migraineurs should preferably be identified by a diagnostic headache diary. The treatment of migraine differs considerably from that of tension-type headache and it is important to educate patients to differentiate between these types of headaches in order to select the right treatment and to prevent medication-overuse headache.

2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness:

Diagnostic criteria include: (A) Episodes fulfilling criteria A-E for 2.2 Frequent episodic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.2.2 Frequent episodic tension-type headache not associated with pericranial tenderness:

Diagnostic criteria include: (A) Episodes fulfilling criteria A-E for 2.2 Frequent episodic tension-type headache (B) No increased pericranial tenderness

2.3 Chronic tension-type headache

This is a disorder evolving from episodic tension-type headache, with daily or very frequent episodes of headache lasting minutes to days. The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, and it does not worsen with routine physical activity. There may be mild nausea, photophobia or phonophobia. Diagnostic criteria: (A) Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year)1 and fulfilling criteria B-D (B) Headache lasts hours or may be continuous (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity 4. not aggravated by routine physical activity such as walking or climbing stairs (D) Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea, 2. neither moderate or severe nausea nor vomiting, (E) Not attributed to another disorder

When it is uncertain how many attacks fulfil one or other set of criteria it is strongly recommended to use a diagnostic headache diary prospectively. In many uncertain cases there is overuse of medication. When this fulfils criterion B for any of the subforms of 8.2 Medication-overuse headache, the default rule is to code for 2.4.3 Probable chronic tension-type headache plus 8.2.8 Probable medication-overuse headache. When these criteria are still fulfilled 2 months after medication overuse has ceased, 2.3 Chronic tension-type headache should be diagnosed and 8.2.8 Probable medication-overuse headache discarded. It should be remembered that some patients with chronic tension-type headache develop migraine-like features if they have severe pain and , conversely, some migraine patients develop increasingly frequent tension-type-like interval headaches, the nature of which remains unclear.

2.3.1 Chronic tension-type headache associated with pericranial tenderness: Diagnostic criteria: (A) Headache fulfilling criteria A-E for 2.3 Chronic tension-type headache (B) Increased pericranial tenderness on manual palpation

2.3.2 Chronic tension-type headache not associated with pericranial tenderness: Diagnostic criteria: (A) Headache fulfilling criteria A-E for 2.3 Chronic tension-type headache (B) No increased pericranial tenderness

2.4 Probable tension-type headache

Patients meeting one of these sets of criteria may also meet the criteria for one of the subforms of 1.6 Probable migraine.

2.4.1 Probable infrequent episodic tension-type headache: Diagnostic criteria: (A) Episodes fulfilling all but one of criteria A-D for 2.1 Infrequent episodic tension-type headache (B) Episodes do not fulfil criteria for 1.1 Migraine without aura (C) Not attributed to another disorder.

2.4.2 Probable frequent episodic tension-type headache: Diagnostic criteria: (A) Episodes fulfilling all but one of criteria A-D for 2.2 Frequent episodic tension-type headache (B) Episodes do not fulfil criteria for 1.1 Migraine without aura (C) Not attributed to another disorder.

2.4.3 Probable chronic tension-type headache: Diagnostic criteria: (A) Headache occurring on ≥15 days per month on average for >3 months (≥180 days per year) and fulfilling criteria B-D, (B) Headache lasts hours or may be continuous (C) Headache has at least two of the following characteristics: 1. bilateral location, 2. pressing/tightening (non-pulsating) quality, 3. mild or moderate intensity, 4.not aggravated by routine physical activity such as walking or climbing

stairs. (D) Both of the following: 1. no more than one of photophobia, phonophobia or mild nausea

2. neither moderate or severe nausea nor vomiting (E) Not attributed to another disorder but there is, or has been within the last 2 months, medication overuse fulfilling criterion B for any of the subforms of 8.2 Medication-overuse headache

IHC CLASSIFICATION –ICHD -2

- 3.1 Cluster headache
  - 3.1.1 Episodic cluster headache
  - 3.1.2 Chronic cluster headache
- 3.2 Paroxysmal hemicrania
  - 3.2.1 Episodic paroxysmal hemicrania
  - 3.2.2 Chronic paroxysmal hemicrania (CPH)
- 3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
- 3.4 Probable trigeminal autonomic cephalalgia
  - 3.4.1 Probable cluster headache
  - 3.4.2 Probable paroxysmal hemicrania
  - 3.4.3 Probable SUNCT

IHS DIAGNOSTIC CRITERIA

3.1 Cluster headache

This entity presents with attacks of severe, strictly unilateral pain which is orbital, supraorbital, temporal or in any combination, lasting 15-180 minutes, occurring from once every other day to 8 times a day. The attacks are associated with one or more of the following, all of which are ipsilateral: conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis, eyelid oedema. Most patients are restless or agitated during an attack. Diagnostic criteria:

A. At least 5 attacks fulfilling criteria B-D

B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15-180 minutes if untreated

C. Headache is accompanied by at least one of the following:

1) ipsilateral conjunctival injection and/or lacrimation, 2) ipsilateral nasal congestion and/or rhinorrhoea, 3) ipsilateral eyelid oedema, 4) ipsilateral forehead and facial sweating, 5) ipsilateral miosis and/or ptosis, 6) a sense of restlessness or agitation

D. Attacks have a frequency from one every other day to 8 per day

E. Not attributed to another disorder

Acute attacks involve activation of the posterior hypothalamic grey matter. Cluster headache may be inherited (autosomal dominant) in about 5% of cases. Attacks usually occur in series (cluster periods) lasting for weeks or months separated by remission periods usually lasting months or years. However, about 10-15% of patients have chronic symptoms without remissions. Pain is maximal orbitally, supraorbitally, temporally or in any combination of these sites, but

may spread to other regions of the head. Pain almost invariably recurs on the same side during an individual cluster period. During the worst attacks, the intensity of pain is excruciating. Patients are usually unable to lie down and characteristically pace the floor. Age at onset is usually 20-40 years. For unknown reasons prevalence is 3-4 times higher in men than in women. Cluster headache with coexistent trigeminal neuralgia (cluster-tic syndrome): Some patients have been described who have both 3.1Cluster headache and 13.1Trigeminal neuralgia. They should receive both diagnoses.

3.1.1 Episodic cluster headache: Cluster headache attacks occurring in periods lasting 7 days to 1 year separated by pain-free periods lasting 1 month or longer. Diagnostic criteria: (A) Attacks fulfilling criteria A-E for 3.1Cluster headache. (B) At least two cluster periods lasting 7-365 days1 and separated by pain-free remission periods of ≥1 month.

3.1.2 Chronic cluster headache: Cluster headache attacks occurring for more than 1 year without remission or with remissions lasting less than 1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-E for 3.1 Cluster headache, (B) Attacks recur over >1 year without remission periods or with remission periods lasting <1 month. Chronic cluster headache may arise de novo (previously referred to as primary chronic cluster headache) or evolve from the episodic subtype (previously referred to as secondary chronic cluster headache).

3.2 Paroxysmal hemicranias They are shorter-lasting, more frequent, occur more commonly in females and respond absolutely to indomethacin. Diagnostic criteria: (A) At least 20 attacks fulfilling criteria B-D, (B) Attacks of severe unilateral orbital, supraorbital or temporal pain lasting 2-30 minutes, (C) Headache is accompanied by at least one of the following: 1. ipsilateral conjunctival injection and/or lacrimation, 2. ipsilateral nasal congestion and/or rhinorrhoea, 3. ipsilateral eyelid oedema, 4. ipsilateral forehead and facial sweating, 5. ipsilateral

miosis and/or ptosis; (D) Attacks have a frequency above 5 per day for more than half of the time, although periods with lower frequency may occur; (E) Attacks are prevented completely by therapeutic doses of indomethacin (F) Not attributed to another disorder. There is no male predominance. Onset is usually in adulthood, although childhood cases are reported. *Paroxysmal hemicrania with coexistent trigeminal neuralgia:* Patients who fulfil criteria for both 3.2 Paroxysmal hemicrania and 13.1 Trigeminal neuralgia should receive both diagnoses. The importance of this observation is that both conditions require treatment. The pathophysiological significance of the association is not yet clear.

3.2.1 Episodic paroxysmal hemicrania: Attacks of paroxysmal hemicrania occurring in periods lasting 7 days to 1 year separated by pain-free periods lasting ≥1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-F for 3.2 Paroxysmal hemicrania (B) At least two attack periods lasting 7-365 days and separated by pain-free remission periods of ≥1 month

3.2.2 Chronic paroxysmal hemicrania (CPH): Attacks of paroxysmal hemicrania occurring for >1 year without remission or with remissions lasting <1 month. Diagnostic criteria: (A) Attacks fulfilling criteria A-F for 3.2 Paroxysmal hemicrania Attacks recur over >1 year without remission periods or with remission periods lasting <1 month

3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT): This syndrome is characterised by short-lasting attacks of unilateral pain that are much briefer than those seen in any other TAC and very often accompanied by prominent lacrimation and redness of the ipsilateral eye. Diagnostic criteria: (A) At least 20 attacks fulfilling criteria B-D; (B) Attacks of unilateral orbital, supraorbital or temporal stabbing or pulsating pain lasting 5-240 seconds; (C) Pain is accompanied by ipsilateral conjunctival injection and lacrimation; (D) Attacks occur with a frequency from 3 to 200 per day; (E) Not

attributed to another disorder.Short-lasting Unilateral Neuralgiform headache attacks with cranial Autonomic symptoms (SUNA), is also described. The literature suggests that the most common mimics of SUNCT are lesions in the posterior fossa or involving the pituitary gland. *SUNCT with coexistent trigeminal neuralgia:* Patients have been described in whom there is an overlap between 3.3 SUNCT and 13.1 Trigeminal neuralgia. Such patients should receive both diagnoses. This differentiation is clinically difficult.

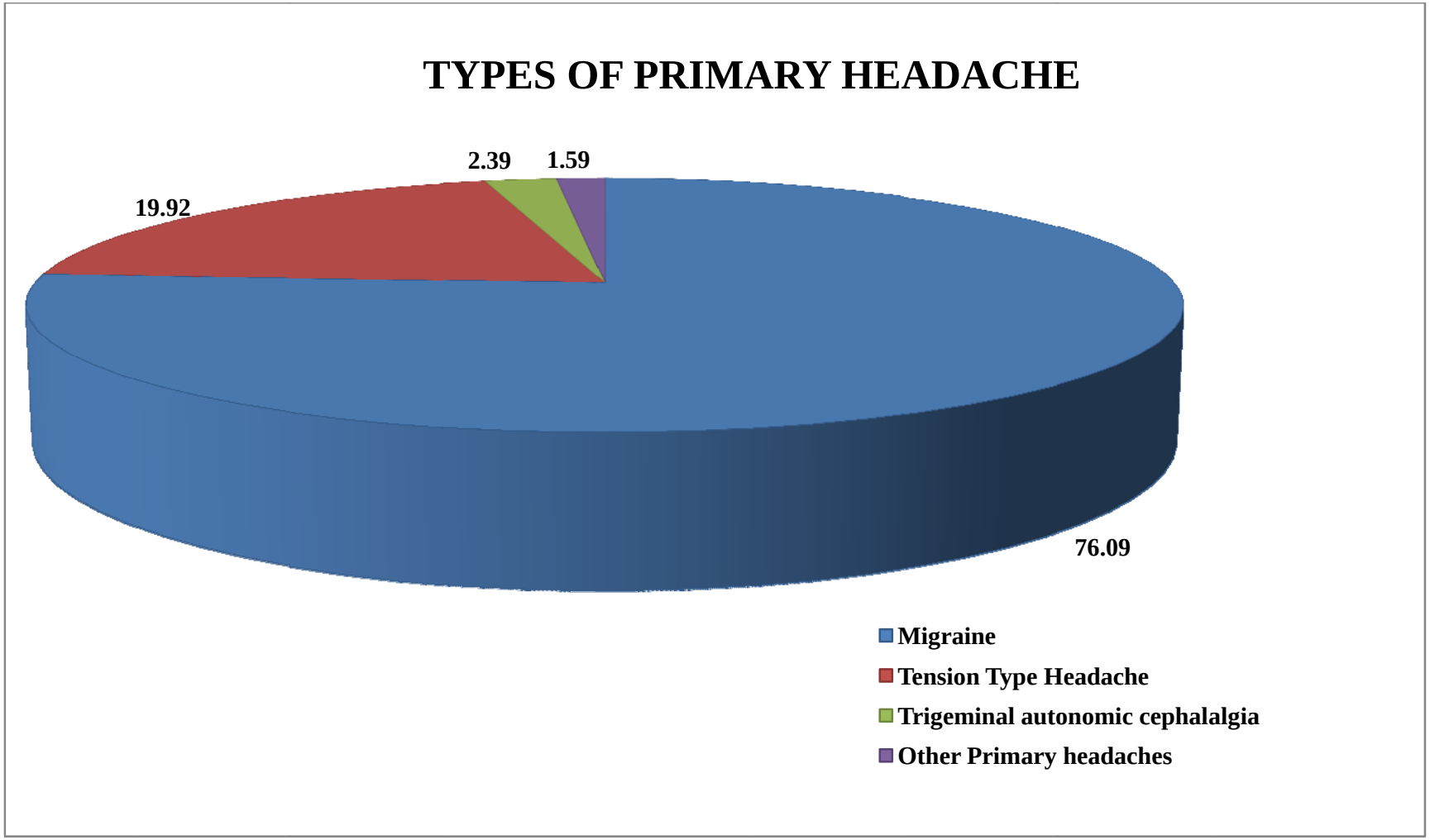
3.4 Probable trigeminal autonomic cephalalgia

Headache attacks that are believed to be a subtype of trigeminal autonomic cephalalgia but which do not quite meet the diagnostic criteria for any of the subtypes described above.

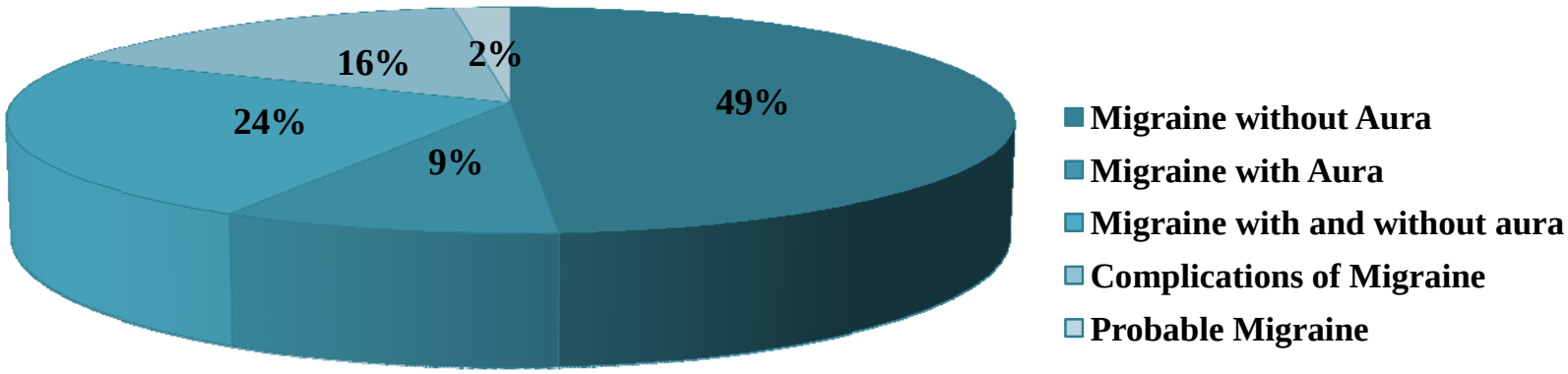
3.4.1 Probable cluster headache: Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-D for 3.1 Cluster headache (B) Not attributed to another disorder

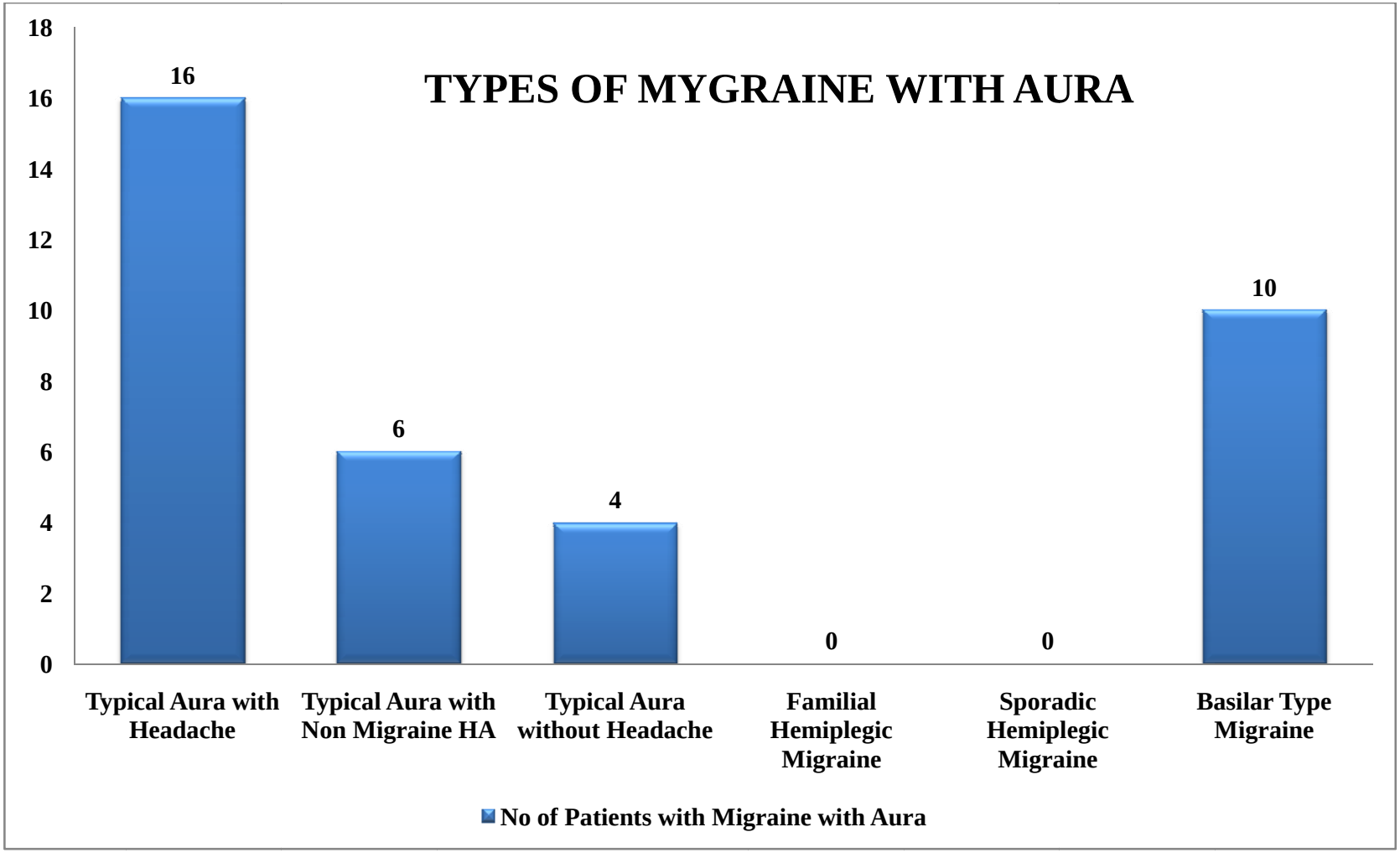
3.4.2 Probable paroxysmal hemicrania: Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-E for 3.2 Paroxysmal hemicrania (B) Not attributed to another disorder

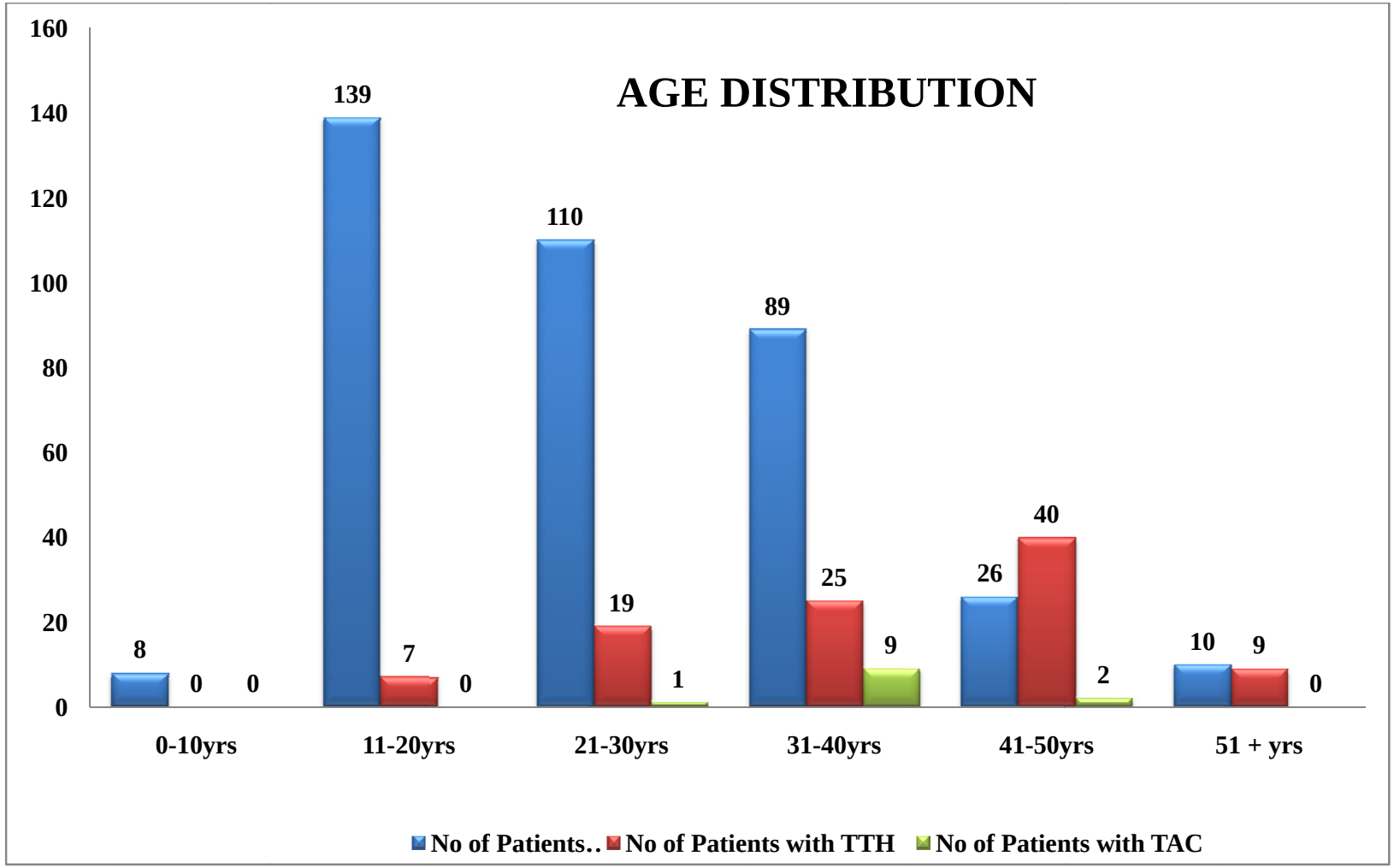
3.4.3 Probable short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing Diagnostic criteria: (A) Attacks fulfilling all but one of criteria A-D for 3.3 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) (B) Not attributed to another disorder

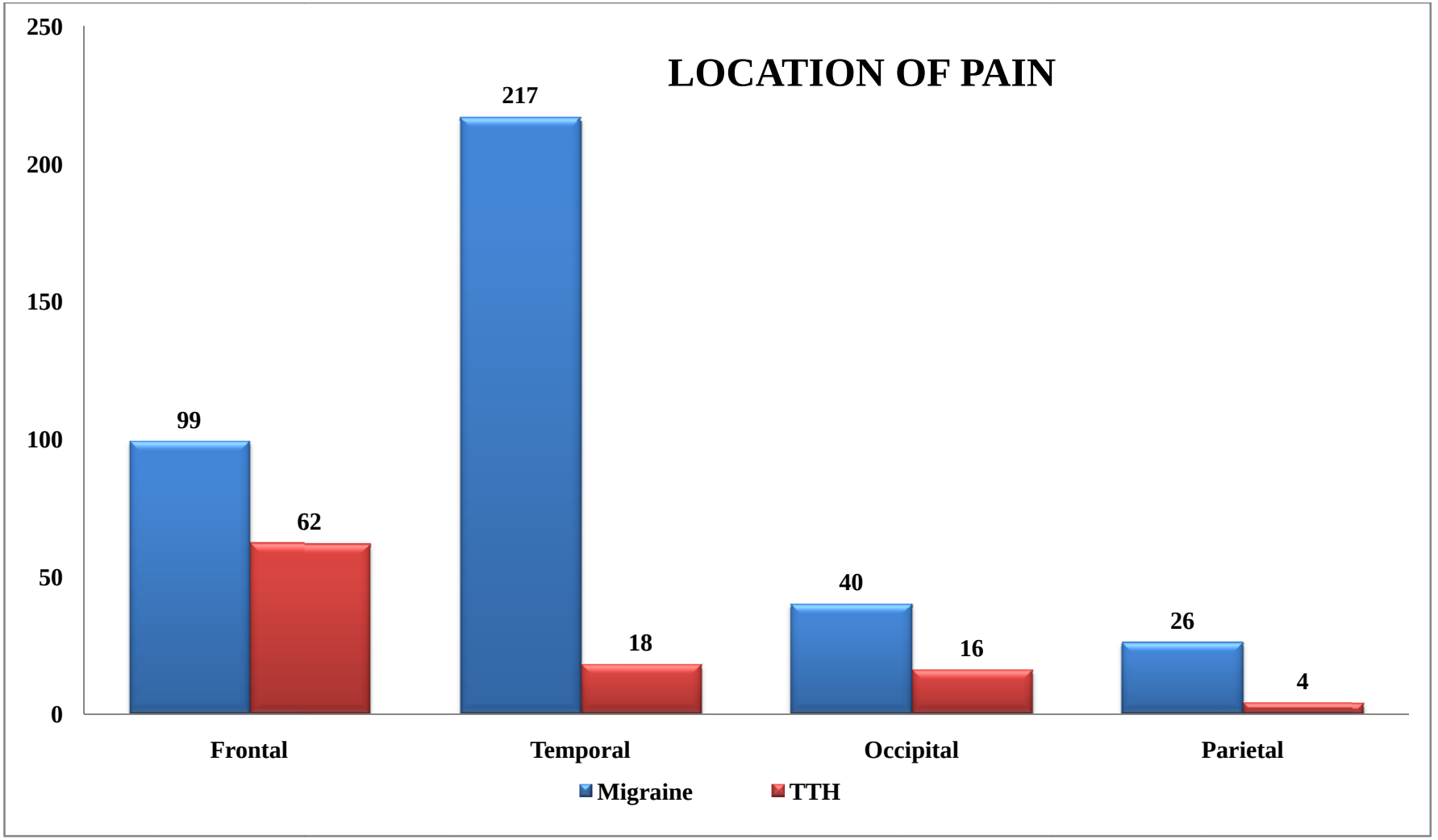

TYPES OF PRIMARY HEADACHE
2.39
1.59
19.92
76.09
Migraine
Tension Type Headache
Trigeminal autonomic cephalalgia
Other Primary headaches

TYPES OF MIGRAINE




TYPES OF MYGRAINE WITH AURA
18
16
14
12
10
8
6
4
2
0
16
6
4
0
0
10
Typical Aura with Headache
Typical Aura with Non Migraine HA
Typical Aura without Headache
Familial Hemiplegic Migraine
Sporadic Hemiplegic Migraine
Basilar Type Migraine
No of Patients with Migraine with Aura


AGE DISTRIBUTION
160
140
120
100
80
60
40
20
0
8
0
0
139
7
0
110
19
1
89
25
9
26
40
2
10
9
0
0-10yrs
11-20yrs
21-30yrs
31-40yrs
41-50yrs
51 + yrs
No of Patients..
No of Patients with TTH
No of Patients with TAC


LOCATION OF PAIN
250
200
150
100
50
0
99
62
217
18
40
16
26
4
Frontal
Temporal
Occipital
Parietal
Migraine
TTH